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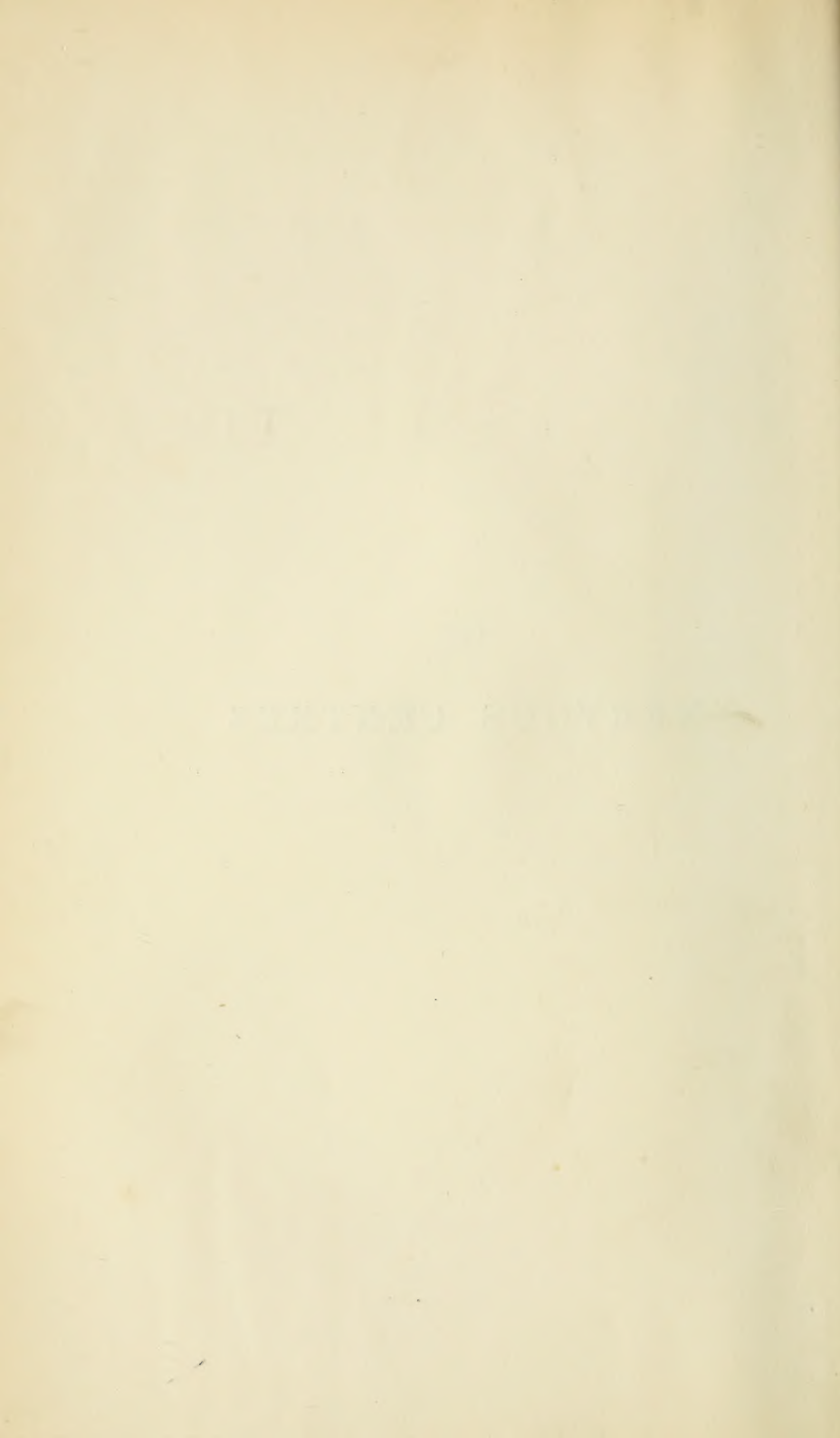
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THE
NERVOUS CENTRES



THE
PATHOLOGICAL ANATOMY
OF THE
NERVOUS CENTRES

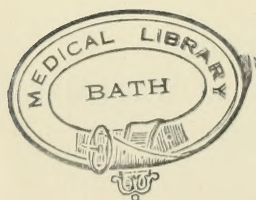
BY
EDWARD LONG FOX, M.D. F.R.C.P.

PHYSICIAN TO THE BRISTOL ROYAL INFIRMARY
LATE LECTURER ON THE PRINCIPLES AND PRACTICE OF MEDICINE AND ON PATHOLOGICAL
ANATOMY AT THE BRISTOL MEDICAL SCHOOL

WITH ILLUSTRATIONS

LONDON
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1874

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TO

MY PUPILS

PAST AND PRESENT

AT THE

Bristol Royal Infirmary and the Bristol Medical School

THESE LECTURES ARE DEDICATED

WITH

MUCH REGARD



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PATHOLOGICAL ANATOMY
OF THE
NERVOUS CENTRES.

INTRODUCTION.

THE following lectures have been given, either as a part of the course on Pathological Anatomy at the Bristol Medical School, or as forming portions of clinical lectures at the Bristol Royal Infirmary.

That anyone giving such lectures should not draw largely from the work of other men would be impossible in a country that has been in the foremost rank in the minute investigation of lesions of the nervous system, among workers of no less note than Maudsley, Bucknill, Reynolds, Radcliffe, Ogle, Allbutt, Jackson, Broadbent, Sanders, Wilks, Lockhart Clarke, Bastian, and many others, and among microscopists such as Dickenson, Herbert Major, and Batty Tuke, and many members of that noble school at the West Riding Asylum under Dr. Crichton Browne.

To these and many others, whose views are quoted

in these lectures, I owe most earnest thanks; and scarcely less am I indebted to some of the French physicians—Trousseau, Jaccoud, Andral, Gintrac, Sandras, and Bourguignon, as well as to Griesinger, Rokitansky, and other German savans.

I have also to express my grateful acknowledgment to Dr. Acland for his permission to cull from Van der Kolk's collection in the Oxford Museum; to Mr. Stirling, of the Edinburgh Anatomical Museum, for much kind help and advice; and to Mr. Charles Berjeau for the interest he has taken in drawing and lithographing the plates.

My desire has been to bring together in a convenient and readable form, for the use of students and practitioners, much that is now scattered in publications of many kinds, as well as some of the pathological experience that has come in my way in the course of some seventeen years of hospital work in a large city. That it is not presented in a better form, that the imperfections are manifold, that the sins of omission, if not of commission, in the full realisation of morbid lesions, are extremely numerous, is due, partly, to the difficulties of writing at all in the midst of a busy professional life, but far more to the vast extent of the subject. In this department of science, as in so many others, we are indeed but as children picking up pebbles on the shore of the ocean of truth.

Still, some points are struggling out of the obscurity of former times, and very much in consequence of the work of the last few years. One is, the tendency to give up the word 'functional' as opposed to

‘organic.’ Probably no organ in the body has been considered to be more frequently the seat of functional disease than the brain. Very many even of the profession at the present day believe that a large number of nervous diseases are associated with no organic change. The difficulty is more of terms than of facts. No doubt many nervous phenomena are met with, and yet nothing is found post mortem that can directly account for them. But this is no proof that lesion has not existed during life. The change in the calibre of the smaller vessels may persist for some time, and yet leave no trace behind, even when nutrition has been for the time materially interfered with. It is only when this interference with nutrition has been extended over a protracted period that so-called organic lesion makes its appearance. But still this variation in the calibre of the vessels is a lesion by itself. Phenomena are not functional because there is no lesion, but because the lesion is transitory and evanescent. It is equally proved by pathological anatomy, and by clinical observation, that no morbid symptom can manifest itself without some alteration in the nutrition of the organ affected.

A second point suggested by this line of work is the close connection between nervous phenomena. Not only are rigor, tremor, spasm, convulsion, mere varieties of the same condition, but they are closely allied to inco-ordination, and through this to paralysis. The same may be said of the connection of excitement, delirium, and mania, with dementia, fatuity, and coma. It would seem as if loss of regulation of

power was only one stage short of loss of power itself.

Many facts await their explanation in the future. Why the same lesion will at one time concentrate itself solely on the posterior columns of the cord, at other affecting only the posterior or the anterior cornua, it is difficult to say ; still more difficult is it to explain the apparent freaks of nature in causing lesion of the trophic nerve fibrils and centres, leaving the motor and sensory tracts untouched, or *vice versâ* ; or its caprice in marking out as its object of attack the vaso-motor nutrition of one muscle, or even of a part of one muscle.

One more consideration is the debt we owe to pathological anatomy for our knowledge of the physiology of the brain and spinal cord. Without for a moment undervaluing the experimental researches of Brown-Séquard on the spinal cord, or the still more important contributions to the physiology of the cerebral convolutions by Hitzig and Ferrier, it must be confessed that nearly all we know of the real functions of the various organs of the encephalon and spinal cord has been drawn from observation of the effects of lesions. Disease is nature's most delicate experiment ; and what knowledge we have of the seat of language in the brain, of the functions of the corpora striata and optic thalami, of the regions specially associated with sensation, with motion, and with thought, is mainly gathered from this, her constant mode of teaching.

In considering the abnormalities of the cerebro-spinal system, the whole class of traumatic lesions has

been omitted. The effects of injuries of various kinds— hæmorrhage, inflammation, &c.—will pass under notice in different sections; but the majority of traumatic lesions are so contained in the regions of pure surgery that they scarcely find a place in lectures devoted to the pathological results of rapid or gradual alterations of nutrition.

It is proposed to divide the subject into two parts: and, first, to describe the pathological anatomy of the brain and spinal cord, and, secondly, the mode in which these pathological results are grouped in certain conditions, which symptomatically have been given special names, as Mania, Melancholia, etc. In pursuing this plan, it will be convenient to divide the first part of the subject into: (1) Congenital Abnormalities of the Cerebro-spinal Centres; (2) Abnormalities of the Vascular System; (3) Inflammations; (4) Degenerations; (5) Tumours; while the second part will include the Pathological Anatomy of Mania, Melancholia, Dementia, Idiocy and Cretinism, General Paralysis of the Insane, Delirium tremens, Paralysis agitans, Epilepsy, Chorea, Hydrophobia, Tetanus, Locomotor ataxy and Progressive Muscular Atrophy, and various local paralyses.

In making this division of the subject, it will be readily understood that it is simply for the sake of convenience. Its imperfections are manifold; specially because in several instances it is logically a cross division. As an example, in the first part, the fifth section, on Tumours, stands by itself, without any connection with the second section, on Abnormalities of the Vascular System. Yet the tumours of the brain and spinal cord

are pathologically divided into those connected with the membranes, those having their origin in the walls of the vessels, and those springing from the neuroglia, the connective tissue of the brain and cord.

No division, however, can be perfect; and it will be attempted in each section to bring into due relations each constituent of the cerebro-spinal system. In the same way also it is not logical in a pathological sense to divide Degenerations from Inflammation, on the one hand, and from some Tumours, on the other. It is not only probable, it is certain, that some forms of degenerations own an inflammatory origin.

It would have been possible to make the division according to the anatomical constituents, and to have taken the organs to pieces, as it were, and thus to have considered in detail the abnormalities of the vessels, of the connective tissue, and of the nerve cells and nerve tubes, that go to make up the structure of the nervous system. But a plan of this kind would necessarily have led to much tautology; a short classification, however, is appended of the lesions to which the various organs within the cranium and spinal column are specially subject.

LESIONS OF THE CEREBRAL DURA MATER.

1. Arrest of development from disease *in utero*.
2. Inflammation (Pachymeningitis).

Thickening. False membranes. Hæmorrhage from new vessels (Hæmatoma). Ulceration. Perforation.

Apparent Hypertrophy. Abnormal adhesion to the bone, or to the subjacent membranes. Abscess.

3. Bony growths. The ossification consisting of true bone.

4. Tumours.

Myxoma.

Lipoma.

Sarcoma.

Fibroma. Fungus of dura mater.

Carcinoma simplex.

Epithelioma.

5. Syphilitic disease affecting the dura mater, and often causing ulceration of this membrane, may be classed under inflammation.

6. Hæmorrhage of dura mater, without previous inflammation—as in purpura, scurvy, injury to the meningeal arteries, perhaps aneurisms.

7. Disease of sinuses. Rupture from distension. Rupture from ulceration. Phlebitis. Thrombosis.

8. Malposition of dura mater, owing to arrest of development of the cranial bones.

LESIONS OF THE CEREBRAL ARACHNOID.

1. Malposition, where a portion of the cranial bones may be absent.

2. Inflammation, acute and chronic. Opacities. Ulcerations.

3. Arachnoid cysts, the result of hæmorrhage.

4. Tumour.

Pacchionian granulations.

Fibroma.

Epithelioma.

Papilloma.

Lipoma, from ependyma of the ventricles.

Cholesteatoma.

Tubercle—probably not primarily growing on the arachnoid.

Hydatids.

LESIONS OF THE CEREBRAL PIA MATER.

1. Malposition, as in meningo-cele and encephalo-cele.

2. Inflammation. Meningitis. Idiopathic, traumatic, syphilitic, alcoholic, epidemic cerebro-spinal, and tuberculous.

3. Fatty degenerations of the walls of the pia mater.
4. Atheroma.
5. Calcareous degeneration, either a sequence of atheroma or of fibroid thickening of the vessels.
6. Cysts on the vessels, especially of the choroid plexus ; these cysts being sometimes filled with a soft material, sometimes with tubercle.
7. Obstruction of vessels by lymph thrown out around and upon them, causing narrowing of their calibre, and often thrombosis.
8. Tubercle, especially affecting the sheath of the vessels primarily.
9. Amyloid degeneration, especially of the choroid plexus.
10. Aneurism, of various sizes, sometimes as large as a bean, more frequently miliary and multiple.
11. Embolism.
12. Rupture from embolism, aneurism, fatty and calcareous degeneration, or sclerosis.
13. Carcinoma simplex.
14. Pearl cancer, often found covered upon its surface by the arachnoid.
15. Epithelioma myxomatodes psammosum.
16. Papilloma of the pia mater.
17. Papilloma myxomatodes.
18. The syphilitic gumma, occasionally attacking the pia mater.
19. Osseous tumour.
20. Œdema.

LESIONS OF THE BRAIN ETC.

1. Malformation.
2. Malposition, as in encephalocele.
3. Anæmia.
4. Congestion.
5. All the vascular lesions found in the pia mater.
6. Hæmorrhages in various parts.
7. Inflammation.
8. Softening.

9. Sclerosis of various kinds and degrees. The so-called hypertrophy of the brain is due to increase of connective tissue.

10. Tumours.

Syphilitic gumma.

Tubercle.

Cholesteatoma.

Fibrous and fibro-plastic tumours.

Carcinoma cerebri simplex.

Epithelioma.

Glioma.

Myxoma.

Psammoma.

11. Parasites.

12. Minute changes of cells of grey matter.

LESIONS OF THE SPINAL MEMBRANES AND CORD.

Dura Mater. Inflammation. Cancer. Fibroma. Sarcoma.

Arachnoid. Inflammation. Spina bifida. Bony plates. Fibrous growths. Possibly tubercle. Cancer spreading from parts contiguous.

Pia Mater. Inflammation. Hæmorrhage. Occasionally tubercle.

The Spinal Cord:

Malposition, as in some cases of Spina bifida.

Malformation. Congenital absence of some portion of cord.

Enlargement of central canal, congenital or otherwise.

Inflammation. Abscess rare, except from the breaking down of a scrofulous mass.

Atrophy.

Softening.

Grey degeneration.

Sclerosis.

Tubercle.

Cancer, probably spreading inwards from contiguous parts.

LECTURE I.

CONGENITAL ABNORMALITIES.

THESE abnormalities may strike the eye at once or may only be discovered by dissection; and depend either on arrest of fœtal development or on intra-uterine disease of the fœtus.

1. We may find the union of two heads into one or two distinct bodies. The junction is commonly an anterior one, but may be lateral. The two chests and the anterior portions of the abdomen as far as the umbilicus may participate in the union. The posterior portion of the head may be double, while the anterior is single. This abnormality may be divided into several varieties, in proportion to the development of the second face. Thus you have, as the first variety, two perfect faces opposed; as the second, an entire face on one side, and one ear, or both ears and one eye, on the other; as the third, an entire face on one side, and one ear or both ears on the other, but without any trace of an eye; as the fourth, an entire face on one side, and no vestige, or hardly any vestige, of any organ on the other side.

2. Two distinct heads may exist on one body, or on two bodies united in the greater part of their extent.

These heads are generally, but not always, of equal size. They sometimes both spring from one neck.

3. There may be a complete absence of the head. When this is the case, there is generally an absence of some other portion of the body. Thus some of the limbs are often wanting, and the vertebral column is very rarely perfect. The two upper vertebræ may be wanting, or the column may be wanting, or the column may begin at the 4th cervical vertebra, or at the 7th, or at the 1st dorsal or the 8th dorsal; and instances are even not unknown where the vertebral column has been absent down to the level of the 10th dorsal, and even to the 2nd lumbar vertebra.

A slight deviation from this abnormality is seen where the head is not really wanting, but is deprived of the greater part of cranium and face.

4. There may be an absence of the brain or of the greater part of the cerebro-spinal system. The shape of the head will depend very much on the extent of deficiency of the cranial bones, or on the amount of distension of the skull by fluid. There are several varieties of this, distinguished by the greater or less extent of deficiency of the cranial contents. Thus the first variety is characterised by the absence of cerebrum, cerebellum, pons, medulla oblongata, and spinal cord. In most of such cases the upper cranial bones and integuments are wanting, and the base is exposed to view, whilst in some the integuments exist and are distended by fluid. The nerves may be present. The second variety is characterised by the absence of cerebrum, cerebellum, pons, medulla oblongata, and part of the spinal cord.

The third, by absence of the cranial contents, whilst the spinal cord is pretty complete.

The fourth, by absence of the cerebrum or cerebellum, whilst the pons, medulla oblongata, and spinal cord are normally developed.

The fifth, by absence of the cerebrum proper only, whilst the cerebellum, pons, medulla oblongata, and spinal cord are normal. This variety may be accompanied by absence of the upper cranial bones, or by their distension by hydrocephalic fluid.

The sixth, which is exceedingly rare, is characterised by the presence of the cerebrum proper, whilst the cerebellum, pons, medulla oblongata, and spinal cord are all absent. These varieties of arrested development seem to depend on physical injuries to the uterus during pregnancy, and sometimes to mental shock experienced by the mother during the same period.

5. Another form of congenital abnormality is characterised by an extraordinary thickening of the meninges, which take the place of, and often imitate, the aspect of brain. This condition sometimes coincides with the abnormality last considered. It may be divided into several varieties, according to the amount of cerebro-spinal centres congenitally deficient.

Thus the first variety shows this thickening of the membranes, with complete absence of cerebrum, cerebellum, pons, medulla oblongata, and spinal cord.

The second is associated with the preservation of the whole spinal cord, whilst the cerebral organs are all wanting.

The third, with preservation of only a portion of the

spinal cord, whilst all the rest of the cerebro-spinal system is absent.

The fourth, with preservation of the spinal cord, medulla oblongata, and pons, whilst the cerebrum and cerebellum are absent.

The fifth, with preservation of the spinal cord, medulla oblongata, pons, and cerebellum, whilst the cerebrum alone is wanting.

The sixth, with preservation of an imperfect brain, whilst the spinal cord is wanting.

6. An uncommon form of congenital imperfection is seen in the cyclocephalous condition, in which there is an approximation or fusion of two eyes in a common orbit. The abnormality is of various degrees, according as the nose and mouth are implicated. It evidently is the result of an arrest of development of the central and anterior parts of the head.

Leaving now, for the present, the abnormalities due to arrest of development, we proceed to those classes of conditions which depend on injuries received or diseases occurring during foetal life. These differ from the preceding in that they are not incompatible with persistence of life.

7. Of these, the most important are those abnormalities which consist in the incomplete, insufficient, or irregular formation of some of the parts of the encephalon. This want of completeness may occur in the membranes, in various portions of the brain proper, the cerebellum, the pons, and the cerebral nerves.

Whenever there is deficiency of structure in the encephalon, there will always be found an increase of

the cerebro-spinal fluid, just as in atrophy occurring in later life.

Ατέλεια of the brain—incompleteness (*atélencéphalie*)—is of very various degrees.

(i) Meningeal incompleteness, in which the membranes may be altogether wanting, or the dura mater may be absent only at the base of the brain, or the falx cerebri may be absent, or may be perforated with holes, the holes representing the areas of incomplete formation; or the tentorium cerebelli may be wholly or partially absent.

(ii) General incompleteness of brain, or such incompleteness affecting several portions of the brain at one and the same time. Such cases have the vault of the cranium thrown back, and the lower jaw very short, whilst the base of the skull is large, the cerebral convolutions almost effaced, and the cerebellum large.

In most of such cases the head is small, and seldom shows its normal dimensions; more frequently it is not only less large than usual, but also flattened on both sides, or on one side, or in front, or at the occiput, and presents various and irregular forms.

With this microcephalous state are found other conditions: manifestations of atrophy, or of a sub-inflammatory process; occlusion of the fontanelles; thickening of the cranial bones; more or less prominent projections of their internal surface; thickening, and a very white tint of the meninges; and collections of serum on the surface and in the interior of the brain.

The brain is reduced in volume ; the convolutions small, or, if large, few in number, or interrupted at some points, and replaced by an accumulation of serum, enveloped by the arachnoid and pia mater, or in a cyst. Various parts of the brain are scarcely or imperfectly developed, or of abnormal consistence ; the septum and pineal gland may be wanting.

The ancient Aztecs afford perhaps the best examples of this microcephalaic condition. Dr. Prichard says : ‘The portraits of the ancient Aztecs, as Humboldt has observed, and some of their divinities, are remarkable for the depression of their forehead, giving a small facial angle ; and this is a form which seems to have entered into the beau-ideal of the race, and which many other American nations imitate by artificial compression of the head.

(iii) Incompleteness of the central portions of the brain. The corpus callosum, the septum lucidum, and the fornix, are sometimes absent.

(iv) Incompleteness of the lateral portions of the brain ; sometimes of one hemisphere, sometimes of the other, partial or entire. The mode of lesion generally consists in a sort of lacuna, a loss of substance, affecting one convolution, or several together. Perhaps loss of substance is hardly the right expression, as a part cannot be lost which has never existed. The lacuna is really formed by congenital want of development of the part. The affected part appears hollowed into a cavity, containing serum, one of the walls of which cavity is formed by the meninges. From this arrangement there result ampullæ, or cysts ; or, when

the lesion is more considerable, sacs, or large bags, taking the place of almost all the hemisphere. In some cases the neighbouring ventricle has been opened. The eroded surface is irregular, the grey matter absent, the white matter sometimes indurated.

There is often serum in the ventricles, with incomplete development of the corpus striatum, optic thalamus, cornu ammonis, mamillary tubercle, crus cerebri, optic and olfactory nerves on the affected side.

A similar loss of substance is seen on the same line of the pons, corpora quadrigemina, and pyramid of the same side; but below the crossing of the nerve fibres in the medulla oblongata the difference in volume is seen on the opposite side. A very remarkable instance of this imperfection of one side of the brain proper is recorded by Van der Kolk. The case was that of an idiot girl, who from her earliest infancy had been paralysed on the right side, but had lived for twenty-seven years. The left hemisphere was much smaller than the right, and the arachnoid over the entire surface was much thickened, and between it and the pia mater a considerable quantity of fluid had collected. The convolutions were much thinner than those of the other hemisphere; the left ventricle larger than the right, containing a good deal of fluid, and the walls of the left ventricle were much thinner. The right corpus striatum was larger than the left, but was somewhat narrower, and the entire body was as if curved round the shortened thalamus.

The difference between the thalami was particularly remarkable. The tænia between the corpus

striatum and thalamus appeared swollen and thickened, in consequence of the thickening of the pia mater lying on it. At the base of the brain the difference between the two hemispheres was especially remarkable, and was in fact so considerable that the length of the right hemisphere exceeded that of the left by twenty-seven millimetres. The left inferior lobe, in particular, was very much atrophied, and its walls were very thin; the convolutions here were very small or had nearly disappeared, perhaps in consequence of the constant pressure of the fluid collected in the horn. The left crus cerebri was thinner, and the left corpus mammillare much smaller, than the right. The left half of the pons mammillare and the left corpus pyramidale presented a much smaller circumference than on the right side; the difference between the corpora olivaria was less. In the cerebellum, on the contrary, the atrophy was on the opposite side, the right hemisphere being in all its measurements much less than the left; consequently the so-called vermis, posteriorly situated in the middle, between the hemispheres of the cerebellum, was drawn obliquely to the right side.

It is extremely remarkable that, while the medulla oblongata above the decussation is larger on the right side, the right corpus pyramidale agreeing with it in this respect, beneath the decussation the atrophy of the left hemisphere had transferred itself to the right side of the spinal cord, as well as of the cerebellum. The atrophy of the right side of the cord was very evident at the level of from the fifth to the seventh cervical vertebra.

In some measure corresponding to this were found the roots (but particularly the ganglia on a level with the brachial plexus) of the fifth, sixth, and seventh cervical nerves on the right side, much diminished in thickness and size; this difference was particularly striking in the fifth and sixth nerves.

There was also found a very remarkable thickening of the bones of the skull on the atrophied side; the anterior and lateral portions of the left side of the skull being much thickened; whilst near the occiput the reverse had taken place, the thickening of the skull in this situation being found on the right side.

(v) Imperfection of the anterior portion of the Brain only.

Both the anterior lobes may be affected. Sometimes the abnormality seems only a very partial arrest of development; at other times the loss is more considerable, and the lobes seem, as it were, truncated.

(vi) Imperfections of the Cerebellum.

One case only has been recorded of the almost complete absence of the cerebellum, both the lateral lobes and the greater part of the central region being found wanting.

More often the arrest of development has only been carried to one lobe, causing simple inequality of the two sides. Sometimes there is found a general smallness of the whole organ.

There is no rule as to the relation of other portions of the encephalon to an imperfect cerebellum. Sometimes the cerebral hemisphere of the same side extends farther back than usual, as though to fill up the space

left vacant by the lateral imperfection of the cerebellum. Sometimes atrophy of one side of the cerebellum co-exists with a similar condition of the cerebral hemisphere of the same side.

At other times the absence of one lobe of the cerebellum coincides with relative smallness of the corpus striatum and olivary body of the opposite side, or with a less development of the pons varolii on the same side as the cerebellar atrophy.

In Van der Kolk's case, the atrophy of the cerebellum was found on the opposite side to that of the cerebral hemisphere.

(vii) Imperfection of the Pons and Medulla oblongata.

Congenital alteration of these parts consists, if life is preserved, only in some modification of form and partial diminution in volume. Entire absence of one side of the pons or of the medulla oblongata is probably unknown, except in anencephalous monsters, stillborn.

(viii) Imperfections of the Cerebral Nerves.—Some few instances have been observed. Thus, when there is no grave alteration of the brain, the olfactory nerves are sometimes wanting. If the two anterior lobes of the brain are fused together, they may be absent; and they are sometimes not found in cyclopic individuals. In the same way, the optic nerves are sometimes absent, whilst at other times the tubercula quadrigemina are not developed.

Although the expression 'arrest of development' has been used more than once in describing varieties of

this incompleteness, it must be generally understood to depend either on injury to the fœtus or on some disease occurring during fœtal life. The brain, being formed late in the order of development, is most likely to suffer from any injury to or diseases of the fœtus, and the pons and the medulla oblongata will probably suffer next in order.

8. *Congenital Hydrocephalus*.—The cases are very rare in which, before birth, fluid accumulates in any portions of the brain or its membranes. Still a few cases are recorded in which this seems to have taken place. Some even of these few are probably due to excessive development of the cerebro-spinal fluid, under conditions of more or less atrophy of some neighbouring portion of the brain itself. The recorded cases must be arranged according to the position in which the fluid is found. Thus :

(i) Fluid has been found between the pericranium and dura mater. This is exceedingly rare.

(ii) *Congenital Hydrocephalus of the Meninges*.—Fluid may exist in the cavity of the arachnoid. This also is very rare, although some cases of it are on record. It has been often mistaken for fluid in the cerebral ventricles in those cases where the layer of nervous substance of the cerebral hemispheres has been thinned to an extreme degree. Under these circumstances, the fluid has been drawn off by tapping, in the full belief that the case was one of external hydrocephalus.

(iii) *Congenital Hydrocephalus of the Ventricles*. Where this occurs without increase in the cranial

volume, it is due to some condition of incompleteness of some portion of the nervous centre, especially with arrest of development of the corpora striata and optic thalami. But it may be attended with increase in the cranial volume, and may be developed either before or after birth.

a. Before birth, very rare.—The fluid may begin to collect as early as the second month of gestation, after a blow on the abdomen of the mother. It may be very chronic, and cases are mentioned of patients in this condition living for 2, 9, 29, 33, 38, and even for 41 years. It is often associated with spina bifida, or with hydrorachis without spina bifida.

b. Developed only after birth.—This may be a true dropsy, depending on disease of the choroid plexus, or it may be a result of inflammation. The brain becomes more or less unfolded, the ventricles distended, the ventricular walls thin, and even almost membranous, sometimes firm and hard, sometimes pulpy. The two lateral ventricles generally communicate freely. The corpus callosum is pushed upward; the septum lucidum may be entirely effaced, although it is sometimes firm; the fornix is thinned and flattened out; the eminences of the ventricles are pressed down. The ependyma lining the enlarged cavities becomes sensibly thickened, of a certain consistence, and vascular, and capable of being peeled off in thick roughish flakes; it is sometimes covered with a thick mucous matter. One ventricle may be more distended than the other.

9. *A condition in which a considerable portion of*

the Brain is situated outside the Cranium.—The characteristics of this abnormality are, that the part of the brain displaced is considerable—that the cranium presents great changes of capacity, form, and constitution, in relation with those that the encephalon has undergone—that life cannot be maintained (or only for a very brief period) with vices of organisation which in their severity are closely allied to those which obtain in anencephalous heads.

According to the direction the brain takes, this abnormality may be divided into : (1) Frontal, in which a portion of the frontal bone alone is wanting ; (2) sincipital, in which the parietal bones may be wholly absent, and a part of the frontal, occipital, and temporal also ; and (3) occipital, in which the exit of the brain takes place in one of three ways—either the issue of the brain is through the posterior fontanelle, or by the absence of the upper and middle part of the occipital, or else the issue of the brain, and generally of the cerebellum, takes place by a large opening at the middle part of the occiput, which often comprises the foramen magnum of this bone, or else the brain, cerebellum, and medulla oblongata are found posterior to the skull, having escaped by a large opening in the posterior part of the head and cervical region of the spine.

Encephalocoele is really only a variety of the abnormality described above. In this condition there is a displacement of a part, more or less circumscribed, of the encephalon across an abnormal opening in the walls of the cranium. This opening may be frontal, sincipital, temporal, or occipital, and of these the occiput is the

most frequent seat, and the frontal next. It does not necessarily lead to early death.

Precisely the same condition may be produced by an opening formed by a wound, or by destruction of some portion of the cranium by syphilis ; but it is also a congenital abnormality. Another variety is encephalocele in a hydrocephalous subject. Under these circumstances, the fluid distending the ventricles is included in the external tumour.

According to the situation of the opening, this abnormality may be frontal, fronto-nasal, sincipital, parietal, occipital, and, lastly, sphenoidal. The latter variety is caused by a large perforation of the sphenoid bone, through which the pituitary body may descend, and project into the cavity of the mouth.

This abnormality seems to be caused by one or other of the following circumstances : either violence to the abdomen of the mother towards the middle period of pregnancy, or inflammation of the brain or the membranes in the fœtus, or compression exercised on the neck of the fœtus by the umbilical cord, by an adhesion contracted with the placenta or the membranes. A still more rare variety is hydromeningocele, in which the dura mater and the serous layer which lines it are lifted up, pressed by the accumulated subarachnoid fluid ; whilst one of the points of the cranial walls, incompletely organised, yields and allows itself to be distended. In this way there may result a tumour very analogous to the former variety, but it differs essentially in the absence of all cerebral expansion unfolded in the hernial sac, or presenting itself at the

abnormal orifice of the cranium. The brain is wholly contained within its normal cranial limits. Sometimes the fluid is from the arachnoid, but this is extremely rare. More often it comes from the ventricle having forced an artificial passage for itself through the thinned layer of cerebral convolutions. The seat of this abnormality is generally the occiput, and its origin in the foetus seems to date from the beginning or middle period of gestation.

We proceed now to the abnormalities affecting the cord itself, some of which may exist alone, whilst others are usually associated with some similar condition of the encephalus.

10. *Absence of Spinal Cord*.—Complete absence of spinal cord is rare, except in anencephalous subjects. It is not compatible with life.

11. *Congenital imperfection of the Cord: incompleteness of Cord corresponding to the same condition affecting the Brain*.—The length, breadth, and form of the cord may vary. The central canal may be dilated: it may be capable of holding a crowquill down the whole length of the cord, and this dilatation, though a structural imperfection, does not seem to give rise to any morbid phenomena. In connection with an absence of the limbs at birth, the cervical and lumbar swelling may be absent. Inequalities of volume are also found in the lateral parts of the cord.

12. *Spina bifida*.—Spina bifida is caused by the division or congenital absence of one or several posterior arches of the vertebræ or the sacrum, allowing the

spinal meninges (which are pressed upon from without by serum, and find no spinal wall to hinder their tendency to bulge consequent on this pressure) to make a projection of greater or less extent. This abnormality is analogous with hydromeningocele. In both we find arrest of ossification, in both accumulation of serum, in both hernia of the meninges. It is found much more frequently in some countries than in others. Countries more or less circumscribed show the largest number of cases in a short time—possibly as a result of close intermarriage. In Holland it is specially prevalent.

Several cases may occur in the same family; and the state of health of the mother seems to have a determining effect, especially as to scrofula, syphilis, &c. The female sex is more liable to it than the male, and it is often coincident with hydrocephalus. Its seat is especially the lumbar regions, and next to this the sacral. It seldom appears in the dorsal and cervical regions, but it has been known to exist at the level of the last cervical vertebra, and to extend thence to the last dorsal. It has also been seen at the occiput, the atlas, and the axis, and sometimes is found in two regions in the same individuals. It is of variable volume, and its form corresponds to the number of vertebræ involved. It is generally soft, but not quite always. It projects more when the child cries.

The colour of the tumour varies: it may be reddish violet, pale or livid, opaque, or semi-transparent. It is generally covered with the skin more or less altered, and sometimes by a thin membrane. The

skin covering it may show various abnormalities, depending on its often being more or less inflamed.

The dura mater always forms an essential element in the tumour. The spinal cord is often seen at the bottom of the cavity, sometimes unaltered, sometimes thin and soft. The extent to which it is implicated in the disease seems to vary according to the position of the tumour. The spinal cord is much more apt to be involved if the disease is higher than the lumbar regions. In spina bifida, the spinal cord often extends lower down than usual, and may enter the sacral canal. Even in the lumbar region, however, it may project into the tumour. When implicated, it does not lie free, but is bound down by adhesions. The nerves of the cauda equina are often found in the tumour. The disease is due to arrest in the development of bone. It is most frequently found in the lumbar region, because ossification here is slower than in other parts of the spinal column. This arrest of development is probably due to local inflammation early in foetal life, which causes points of adhesion between the walls of the spinal canal, meninges, and cord, and provokes hypersecretion of serum.

The fluid may accumulate in the central canal of the cord, in the sac of the arachnoid, or between the pia mater and arachnoid.

Consult on this subject—

Gintrac, 'Maladies de l'Appareil nerveux.'

Holmes, 'St. George's Hosp. Rep.,' vol. i.

Prichard, 'Natural Hist. of Man.'

Van der Kolk, 'New Syd. Soc.,' vol. xi.

'Journal of Mental Science,' Oct. 1873, p. 361.

'Brit. and For. Med.-Chir. Rev.,' July 1874. 'Microcephalism.'

LECTURE II.

ABNORMALITIES OF THE VASCULAR SYSTEM.

THIS subject includes all diseases of the cerebral vessels, cerebral and spinal hæmorrhages, and that battle-ground of pathologists, the morbid conditions that are said to result from a variation in the amount, and a modification in the course, of the blood in the cerebral vessels. It also closely infringes upon the next subject—inflammation; so much so, that by some observers inflammation is found under this classification as a morbid state of the vessels; and in the case of tuberculous meningitis, in which the original lesion is first found in the tissue absolutely forming part of the vessels themselves, the separation of the disease from those of the vessels is in the highest degree unscientific, although for practical purposes it may be convenient.

I. We take then, first, the diseases of the cerebral vessels; and these form a natural arrangement, according to the kind of vessels implicated—arteries, capillaries, or sinuses and veins.

1. Diseases of the cerebral and spinal arteries.

a. Aneurisms.—These are most frequently found of the basilar, the middle cerebral, and the internal carotid, sometimes within the cavernous sinus, some-

times outside it. But this condition is met with affecting most of the arteries of the brain—the middle meningeal, the anterior cerebral, the anterior communicating, the arteries of the corpus callosum, the posterior communicating, the vertebral, the posterior cerebral, and the cerebellar. It is far more common than is supposed to meet with miliary aneurisms of the brain: miliary aneurisms of the internal vessels of the brain and of the pia mater often co-exist. They constantly co-exist also with aneurisms, small or large, of other vessels of the body; and as instances of their co-existence with aneurism elsewhere may be mentioned the fact that with miliary aneurism of the cerebral vessels has been found a similar condition of the vessels of the hands and feet, shoulders, œsophagus, and stomach. They occur also in the retina.

They seem to be produced quite independently of atheroma, but rather to depend on a peculiar alteration of the tissue of the vessels, and primarily of the inner coat, to which the name ‘Arterio-sclerosis’ has been given. This condition is sometimes found coincident with sclerosis of the spinal cord. MM. Charcot and Ch. Bouchard deny this condition of sclerosis, and say that the inner coats of the arterioles first become ruptured, and thus give rise to the formations of a dissecting aneurism. This state of things may continue for some time; or, in consequence of a process of regression, nothing remains but a little pigmentary tubercle. Lastly, in other cases the adventitious tunic at length becomes ruptured, and gives rise to a cerebral hæmorrhage.

Zenker, however, upholds the view of sclerosis of the inner coat, either at the seat or in the immediate neighbourhood of the aneurism.

Instances are met with of the vessels of the brain, cerebellum, pons, and spinal cord being thus affected at the same time. No age is exempt from this 'premature old age of the arteries': it occurs even in children.

In many people there seems to be a true aneurismal diathesis; and certainly a predisposition to aneurism is influenced by the speciality of the race. It is more common in England than elsewhere, and in men than in women.

Miliary cerebral aneurisms exist in various cachexias, and in some general maladies; and although they may persist for some time without giving rise to any symptom, yet often enough they are accompanied by headache and vertigo; and successive attacks of paralysis, quickly recovered from, afford almost certain proof of their existence in the brain.

They may co-exist with and be more or less caused by cancer, the tuberculous cachexia, in which the retina is sometimes thus affected, Bright's disease, and chronic alcoholism, in which, again, the retina suffers in the same way, even in those cases in which the kidneys have not become diseased. Alcohol, indeed, is a frequent cause of aneurism, as being probably a frequent cause of the arterio-sclerosis just mentioned.

This abnormality of the small vessels of the brain is found also in cases of general paralysis, lead-poisoning, rheumatism, gout, leucocythemia—the capillaries, distended with white corpuscles of the blood, not unfrequently giving way—pregnancy, and purpura.

The larger aneurisms depend on a condition of arteries somewhat different from the arterio-sclerosis. The walls are sometimes thin and transparent; sometimes thick, hard, cartilaginous, encrusted with lime-salts, or spotted with bony plates.

Their effects vary immensely according to their seat. They may compress a nerve, or give rise to a sensation of palpitation in the head that is almost insupportable; or, if aneurism exist within the cavernous sinus, the circulation of the blood may be arrested, the veins of the retina will be dilated, and even exophthalmia be produced.

If rupture occurs in the sac, a sort of arterio-venous aneurism is caused. Serous effusions, both in the arachnoid and in the ventricles, may be the effect of aneurism of the cerebral vessels.

In a man, aged 45, who died of cancer of the stomach, all the arteries of the circle of Willis were very atheromatous. On the left posterior cerebral artery, just as it crosses the crus cerebri, was a small aneurism, the size of a large pea. The crus beneath this was rather softened, and the locus niger exposed.

b. Atheroma.—The interference with the calibre of an artery by atheroma is a common cause of thrombosis. It is practically an arrangement of greyish or yellowish-white patches interposed between the internal and middle coats of the artery. These patches may be dissolved in alkalies and in ether. They constitute a fatty degeneration of the internal and middle coats, perhaps specially of the latter.

c. Cartilaginous, ossaceous, or calcareous state of the

Arteries.—This condition is most common in the arteries of the base, and specially in the principal trunks. It occupies the same position in the arterial wall as atheroma, and indeed is very frequently a further stage of degeneration. It is most common in advanced age, but is found at all ages. In some cases it seems to be a degeneration occurring upon chronic arteritis. The arterial walls are rendered very brittle, and hæmorrhage is a frequent result. Yet the rupture of the artery does not always take place at the most diseased part. The disease interferes not only with the resilience, but actually with the calibre of the vessel, and there results either congestion without hæmorrhage, or such a modification in the course of the blood that great pressure is brought to bear upon some other part of the vessel, and so hæmorrhage. As a further result of the lesser evil—congestion—we find serous effusion into the arachnoid cavity, the pia mater, and the ventricles.

For a statement on hyaline-fibrosis, and hypertrophy of the muscular coat, see Lecture VII.

d. Perforation. — Sometimes perforation occurs without our being able to detect any lesion of the vessel that could have led to it. More often, however, it results from a local alteration of the arterial walls (differing somewhat from arteritis), by which they become thickened and softened, and in some places eroded, ulcerated, and destroyed. Of course we are leaving out of the question perforation from traumatic lesion of any kind, but it may be connected with gradual erosion of the bony walls from syphilitic or other causes.

e. Thrombosis.—Rare in arteries. It is practically a concretion or coagulation of blood contained in the vessels; formed *in situ*, not brought from a distance. It may occur without any appreciable lesion of the arterial walls; and in this case it is from what we are accustomed to call hyperinosis of the blood, an excess of fibrine, as in several acute diseases, and markedly in acute rheumatism. But more generally it seems to depend upon some condition that interferes with the normal calibre of the vessel itself, or which renders the internal coat rough or unequal. This may be from inflammation, but not very frequently. Atheroma is a more common cause.

The thrombi are more dense at their circumference than at the centre. Sometimes they contain small calcareous deposits. Men are more subject to this lesion than women; but old age seems not to be more liable than the other periods of life. The internal carotid, the middle cerebral, the basilar, and the vertebral arteries are most commonly affected.

f. Embolism.—Due to a coagulum brought from a distance, and not formed *in situ*. It may be brought from the heart itself, or its valves, from the walls of the aorta, or from the cavity of an aneurism.

Several arteries may be obstructed at one and the same time; sometimes all on one side, at other times some arteries of one side of the brain and some of the other.

We may meet with embolism obstructing the main trunk of an important artery, causing perhaps sudden aphasia, or hemiplegia; or the main trunks may escape,

and the smallest branches be thus blocked, as they have been found to be in the corpus striatum in chorea.

The left middle cerebral artery is the most frequent seat of embolism, and next the right Sylvian artery. It is a constant cause of hæmorrhage. The female sex is much more liable to embolism than the male; and youth and adult age more than other periods of life.

2. *Diseases of the Capillaries.*—The cerebral capillaries do not seem to be affected with aneurism. Even the smallest miliary aneurisms are connected with the arteries, and do not attack the capillaries. But these latter vessels may be dilated from various causes, sometimes from backward pressure of blood, owing to venous obstruction, and sometimes from the augmented *vis a tergo* due to hypertrophy of the heart.

They are also liable to atheroma, though less so than the arteries, and, probably as a consequence of atheroma, to calcareous infiltration.

When the calibre of the capillary vessel is partially interfered with and obstructed by atheroma or calcareous degeneration, a thrombus may form in it; but embolism of the capillaries is rare. Blandford and Charlton Bastian, however, mention it. The so-called capillary hæmorrhages are generally due to rupture of the smallest arteries. Sometimes, however, rupture of the capillaries takes place, especially in those minute vessels that often encircle an old hæmorrhage in the brain, or a spot of softening.

3. *Diseases of the Cerebro-spinal Venous System.*—
a. Inflammation of the Cerebral Veins and Sinuses of

the Dura Mater.—Inflammation of the veins and sinuses generally co-exist. The vascular walls lose their transparency, and become thick, rugose, soft, and reddened. The vein or sinus attacked contains clots of various colours and consistence, according to their age. These clots form true thrombi, and inflammation of the veins and sinuses is a not unfrequent cause of cerebral thrombosis; they also contain pus, false membranes, &c.

This inflammation may depend on various causes. Damp, scrofula, alcohol, misery, all predispose to it; whilst accidents, ulcers, or abscesses of the scalp, anthrax of the neck, pus in the pleural cavities, and scarlatina, are all exciting causes. But perhaps the most common cause of inflammation of the sinuses, and more frequently than is supposed of the cerebral veins themselves, is inflammation of the internal ear and the pars petrosa. Under these circumstances the lateral sinus is the one most usually affected: the dura mater is often raised by pus, becomes thick, spongy, grey or black, and ulcerates. The corresponding lobes of the brain are covered with pus, or with brown, grey, or slate-coloured matter. There is effusion of pus or serum under the arachnoid, or in its cavity, or in the ventricles. The cerebral substance is injected, or studded with small clots or with larger hæmorrhages, and in many cases softened. When the cerebral veins are likewise implicated, there is found also cerebral abscess. Secondary abscesses of the lungs, liver, spleen, and kidneys may be met with.

b. Thrombosis.—Sometimes the result of phlebitis, as

above stated. It may arise from other causes, especially from anything that forms an obstacle to the circulation of the blood in the brain, such as tumours, preventing the return of blood from the head; probably, also, any condition that leads to partial asphyxia, such as asthma. It is most common in early life. The vein is seldom attacked alone: it is associated with thrombus of a neighbouring sinus, on the one hand, or more commonly with thrombi in the capillaries and small artery with which it is connected. The thrombus is occasionally found partially organised.

As a result of venous thrombosis, we may meet with serum in the cavity of the arachnoid in some cases, or a hyperæmic condition of the pia mater, or sometimes with small hæmorrhages on the surface of the cerebral hemispheres or within them. The brain is sometimes healthy, sometimes more or less softened in the neighbourhood of the affected vein.

Th. von Dusch collected fifty-seven cases in which the formation of the thrombi in the sinuses of the brain is mentioned. A comparison of these cases in reference to their causation shows that in thirty-two the thrombosis was the result of gangrenous, erysipela-tous, and suppurative inflammations of those parts of the body (neck, face, orbits, bones of the skull, brain and its membranes) whose vessels are in close connection with the sinuses. In four it appeared to result from a diminution of the calibre of the sinus, by the intrusion of foreign bodies and tumours, or from pressure from without upon the sinus, or upon the internal jugular vein. In fifteen it appears to have been occasioned by circumstances which lessened the force of

the circulation, namely, by previous debilitating diseases in individuals for the most part feeble already, as aged persons and children. Lastly, in six cases nothing positive as to the cause could be ascertained from the history.

c. Cartilaginous and osseous conditions of the Cerebral Veins.—This abnormality, which is found in the inferior cava and other veins, is very rare in the cerebral veins, and probably unknown in the sinuses.

d. Rupture of the Cerebral Veins and Sinuses.—Rupture of veins is very rare. At the point of confluence of the sinuses the walls of the sinuses are often much thinned by the constant pressure of the blood arrested in its course from the brain. Under these circumstances, the walls may yield at this point, and venous blood be effused along the base of the brain, and down the spinal canal.

II. HÆMORRHAGES.

1. *Hæmorrhage outside the Brain and Spinal Cord.*

a. Between the Cerebral Dura Mater and the Bone.—This depends on fracture of the skull; on blows on the head, without fracture of the skull; on erosion from syphilis; on congestion of the meningeal vessels; on morbid conditions of the blood itself, as scurvy or purpura; and, lastly, Gintrac mentions as a cause of hæmorrhage in this position, vascularity of the Pacchionian glands that have pushed through the dura mater. It is an uncommon condition, except from a traumatic cause.

b. Cerebral Hæmorrhage beneath the Dura Mater, in the cavity of the Arachnoid.—The blood is found in this situation under circumstances varying according to the time at which it has been effused, and also according to the presence or absence of surrounding inflammation. Thus we meet with blood in a liquid state, or partly coagulated, or wholly coagulated, lying in the cavity of the arachnoid, surrounded by the parts in a normal state, without any sign of inflammation; or, on the other hand, we may meet with blood surrounded by a false membrane of various degrees of thickness, sometimes unorganised, sometimes partially organised; and, lastly, it may be surrounded by an old thick cyst wall thoroughly provided with vessels. Hæmorrhage from the under-surface of the dura mater usually occurs as a sequence of inflammation of that organ, the blood coming from the tender vessels of the partially organised effused products.

For further reference to the arachnoid cysts, so formed, see Lecture VII.

c. Hæmorrhage: sub-arachnoid, cerebral.—This may occur either on the convex surface of the brain only, or at the base only, or both at the base and on the convexity together. It is said that sub-arachnoid hæmorrhage at the base of the brain is never met with in the insane. The symptoms are grave, and the prognosis serious in proportion to the extent of the hæmorrhage. Hæmorrhage of the convexity depends most frequently on miliary aneurisms of the pia mater. It is usually very small in amount, forming one or more ecchymoses, and probably then giving rise to

no symptom. At other times, many of the miliary aneurisms are found, and the results of their rupture are seen on the convexity, varying in colour and consistence according to the date of the hæmorrhages. At other times, again, these miliary aneurisms co-exist with one of a larger size, from the rupture of which the whole of the convex surface of the brain is covered with blood. A large hæmorrhage of this kind may, moreover, depend on external injury to the skull, or on scurvy or purpura.

When subarachnoid hemorrhage of the base is met with, it is generally due to fracture of the skull, with rupture of the membranes or laceration of the brain, or both ; or to the giving way of an artery at the base, especially the basilar ; or to the fact that a large cerebral hæmorrhage has partially found its way externally.

Subarachnoid hæmorrhage of the convexity and base together will generally be found, excluding very severe local accidents, to depend on the co-existence of large and miliary aneurisms, a condition, so to speak, of aneurismal diathesis ; but, once again, it may be well to remind you that an aneurism which by its rupture leads to great hæmorrhage of the base need not be of any vessel external to the brain itself.

d. Hæmorrhages of the Spinal Meninges.—This may be outside the spinal dura mater, or in the cavity of the arachnoid or sub-arachnoid. All three forms may be due to external injury to the spine, but they may depend on other causes. The two latter varieties especially may be caused by great muscular efforts,

by venereal excess, perhaps by the abuse of alcohol. Certainly rupture may occur after great congestion, such congestion being induced by suppression of the catamenia, or by interference with respiration. Unless the hæmorrhage be of great amount, much of the clot is rapidly absorbed, but it generally sets up some inflammatory action of the meninges in its neighbourhood, the membrane becoming thickened and opaque; and very often the nervous tissue beneath the seat of effusion is softened. This obtains also in hæmorrhage of the cerebral membranes.

2. *Hæmorrhage of the Brain.*—Hæmorrhage may occur in any portion of the nervous centres, and in several spots at one and the same time. Thus clots of various sizes may be found in the cortical substance of the brain, in one or both of the anterior, middle, and posterior lobes, in one or both of the corpora striata, or optic thalami, in the ventricles, in the cerebellum, the pons, the medulla oblongata, and spinal cord.

(i) *Hæmorrhage of the Cortical Substance.*—Seldom of one spot only, or of very small extent. When it is so, it depends generally on local disease, such as adhesion of the membranes to the part, disease of the pars petrosa, otitis, &c. More frequently it is found in larger amount or in numerous spots, and the blood may be seen mingled with the nerve matter, from local encephalitis or local softening having been set up in the immediate neighbourhood of the clot. These spots may be surrounded by cysts.

At the periphery of the brain, and especially on the convex surface, hæmorrhage of the cortical

substance occurs in a miliary form, 'Capillary apoplexy,' as it has been termed. If no inflammation has been set up around these spots, we find a few globules of blood, or merely some blood-crystals. This form occurs under several conditions, such especially as miliary aneurism, purpuric alteration of the blood, thrombosis of the sinuses or of the veins of the pia mater. Old age is most subject to it.

It is not very unusual to meet with the co-existence of hæmorrhage of the cortical substance and of the meninges. The arteries are sometimes atheromatous, cartilaginous, calcareous, sclerosed, or aneurismal; and Gintrac says that the corpora striata, in cases of hæmorrhage of the cortical substance, are sometimes found injected, softened, of various colours, and pitted with small irregular cavities.

The brain substance is more often injured by hæmorrhage in the middle lobes than in either the anterior or posterior lobes; and the lesion in the middle lobes is of greater gravity, partly because of the near neighbourhood of the corpora striata, partly because of the tendency of the effused blood to find its way into the ventricles.

In the anterior lobes the hæmorrhage is generally circumscribed, but it may open into the lateral ventricles or externally, or in both situations at once. Coincident hæmorrhage may occur in the anterior and some one of the other cerebral lobes, or in the interior of the lobes and the cortical substance, or in the lobes and the meninges, at one and the same time; but hæmorrhage of the corpus callosum alone is extremely rare.

Hæmorrhage of the middle lobes may be met with in one lobe only, and then the gravity of the case will be in direct proportion to the extent of the lesion. Death may occur in a few hours, or not for several years. The lesion may attack both lobes simultaneously or successively; or rupture may take place at the surface of the brain, and the blood originally effused in the middle lobe may find its way into the meshes of the pia mater; or this rupture may take place into a lateral ventricle, causing symptoms that usually are quickly fatal, though cases are on record where life has been prolonged, but probably only where the amount of blood poured out into the ventricle has been small; or, again, this complication may arise not from an extension to the ventricle of the original lesion, but from rupture of a cyst that had formed round a previous clot; or, again, the effused blood may find its way simultaneously into a ventricle and at the surface of the brain.

The posterior lobes are less often affected than the middle, and hæmorrhage there seems to be of far less serious import.

It may be circumscribed in one or both posterior lobes, or it may occur in one of the posterior lobes, and open at the surface of the hemisphere or into one of the ventricles, or simultaneously into one of the ventricles and at the surface of the hemisphere. This hæmorrhage has been known to occur in early childhood, at four years of age. The duration of life may be much greater after hæmorrhage in the posterior than after hæmorrhage in the middle lobes.

Extravasation of blood occurs in the brain in bulk or in a punctated form. This punctated form may depend on one or other of the following causes: Hyperæmia, inflammation, embolism of the small arteries of the brain, internal inflammation of vessels, minute aneurism, and purpura hæmorrhagica. It is seen in the form of many blood-points, arranged very close together, in a very limited area. Each point is surrounded by a delicate capsule, furnished, as Rindfleisch says, by the fibrogenous substance of the circumfluent nutritive fluid. The changes that take place in this minute extravasation are either to yellow softening, or to suppuration, or to direct organisation of the clot. Of this latter process, Rindfleisch's account is, that organisation of the capsule occurs first, and then of its contents. Colourless blood-cells gradually take the place of the red ones, without the capsule being opened at any place, and so this germinal tissue is directly transformed into fibrinous connective tissue. In the capsule itself cleft spaces occur, which divide the capsule into multiple layers. In these spaces connective tissue corpuscles become visible; and, finally, the inner connective tissue fuses completely with the outer, and a small compact nodule is formed.

Bulky hæmorrhage can only find space by displacement of the cerebro-spinal fluid and by emptying the cerebral vessels. If the first injury is withstood, the contraction of the fibrine enables the vessels to fill again, and thus the brain is nourished. The clot becomes encapsulated, and goes through changes as in the punctated form. Part of the blood is changed into

pigment, part becomes connective tissue, and forms the cicatrix ; or, if the process does not go so far, a cyst is formed, filled with clear fluid. This connective tissue growth may not be complete for a long time : round an extravasation of any size it is probably not thoroughly developed before the end of the second year.

(ii) *Hæmorrhage of the Corpora Striata.*—These organs are the most frequent seat of cerebral hæmorrhage, from their special texture, from the abundance of grey matter in them, and from their richness in vessels. The lesion may occur in and be confined to one corpus striatum only, and may either destroy life rapidly, without any attempt at local repair, or not for twelve months, in which case a more or less complete cyst may have formed, the corpus striatum being to a great extent a mingled débris of blood and brain tissue; or life may be prolonged with paralysis for a much longer period, and when death occurs, either from a second hæmorrhage or some other cause, a cyst is found of some thickness, traversed by bands, and causing either a depression or a projection in the corpus striatum, according to the original position of the lesion and the amount of contraction the connective tissue has undergone. Or the hæmorrhage into one corpus striatum may be complicated by rupture of the organ towards the lateral ventricle and extravasation of blood into this cavity—a lesion generally very rapidly fatal, though not necessarily so. The left corpus striatum is more frequently affected in this way than the right; or, again, hæmorrhage may be met with in both corpora striata, these organs being sometimes attacked successively, sometimes simul-

taneously. Hæmorrhage of the neighbouring organs, the optic thalami, is not so common, and the effects are often less marked, the paralysis being less intense, even if there is any diminution of motor power from lesion of these organs alone. The lesion may be circumscribed in the optic thalamus, or may extend into the corresponding cerebral peduncle, or may open at the circumference of the brain, or more frequently may extend and allow extravasation into the neighbouring lateral ventricle; and, lastly, hæmorrhage of one corpus striatum and one optic thalamus may occur simultaneously, sometimes with one spot of lesion common to both, at other times with lesions distinct in each organ. When hæmorrhage occurs into the ventricle to an extent that is not rapidly fatal, the ependyma is generally after a time found a good deal thickened. This lesion is met coincidently with hæmorrhage of other parts of the brain. We find hæmorrhage of an optic thalamus and a corpus striatum with hæmorrhage of the cerebral lobes and of the meninges, or of a cerebral lobe with the meninges intact.

Hæmorrhage of the cerebral ventricles may occur from various morbid conditions of the plexus choroides. The plexus may be more vesicular than usual, with vessels greatly distended with blood, and showing ruptures in several places; or, indeed, engorged from any cause that interferes with the normal flow of blood through these vessels. This lesion is sometimes coincident with a breaking down of the septum lucidum; at other times the ventricular walls are injured, softened, or eroded, either from pressure of the effused

blood upon them, or in consequence of mal-nutrition from impeded blood supply, or from extension of lesion from a corpus striatum or optic thalamus. In very rare cases, blood is found in the ventricles without any lesion that can readily account for it, with healthy plexus choroides and venæ Galeni, with floor, and walls, and roof of the ventricles perfectly healthy. In these cases the hæmorrhage has been supposed to have been due to a kind of exhalation, but of this there is no sufficient proof. It probably, however, depends upon some abnormality of the blood itself, rather than of any vessel. Hæmorrhage of the ventricles may co-exist with hæmorrhage of the corpora striata, optic thalami, cerebral lobes and meninges, or of any of them.

Hæmorrhage of the cerebellum is very rare, and when it occurs it is usually in advanced age. The course of the malady is generally continuous, but some cases are recorded in which there has been marked amelioration. The most rapidly fatal cases are those in which the blood has found its way into the fourth ventricle. Hæmorrhage may occur in the cortical substance, in the right or the left lobe, or in both, or in the central region of the cerebellum; and such lesions may co-exist with similar hæmorrhage in any one or more of the following regions—the meninges, the cerebral lobes, the corpus striatum, the optic thalamus, the cortical substance of the brain, the lateral ventricles, and the pons.

Hæmorrhage of the pons is a very serious condition, and the gravity of the case is again in direct

proportion to the size of the hæmorrhage. It is not very unusual to find two, three, or even four miliary hæmorrhages in the pons, and yet life has been prolonged. In some cases the blood is contained in the pons itself, and the symptoms, of course, vary according to the exact position of the hæmorrhage. The blood, however, may find an exit into the fourth ventricle, or at the inferior surface of the pons itself, or in both directions in the same case; or, again, it may break into the crus cerebri on one side, or both, or into the crura cerebelli.

Hæmorrhage may also occur in one or both of the crura cerebri, and perhaps specially in the locus niger.

The two following cases are instances of hæmorrhage into the pons :—

Woman, aged 73, with purpura. A little dark fluid was smeared on the outside of the right hemisphere. In the right lateral ventricle was a ragged opening in the optic thalamus, from the rupture of a vessel. Almost all the central part of the right hemisphere was broken down by the blood, and the left ventricle was filled with fluid blood, from rupture of the septum. The pons, on transverse section, showed five or six small extravasations in its substance.

Man, aged 45, with purpura. A large clot extended over almost all the convex surface of the right cerebral hemisphere. On making sections of the brain, a small clot was found in the posterior angle of the left lateral ventricle, and one about the size of a nut in the anterior portion of the corpus callosum. A very small clot in

the right optic thalamus. Both corpora striata healthy. A small clot in the posterior portion of the third ventricle, the blood passing through the iter a tertio ad quartum ventriculum, and distending the fourth ventricle, making its way round to the lower portion of the cerebellum. The medulla oblongata was healthy ; but on section of the pons, the fibres of this organ seemed to be laid alternately with horizontal clots, as if all the vessels here had given way.

Hæmorrhage of the medulla oblongata is also rare. Death occurs very rapidly under such circumstances, but even then never quite suddenly. Sudden death, unless induced by certain poisons, as prussic acid, is always due to disease of the heart. No brain lesion, however intense and however rapidly fatal, ever induces an absolutely sudden fatal event.

Hæmorrhage into the fourth ventricle seldom, if ever, results from the breaking through of the blood from the medulla oblongata. Death, in hæmorrhage into this latter organ, is too rapid for that. It commonly arises from blood that has found its way in the ordinary channel from the lateral ventricles, or the third ventricle, or else from the giving way of the substance of the cerebellum or of the pons.

The conditions that precede cerebral hæmorrhage, some of which cause it, whilst others are coincident with it, are :

(1) Blows or wounds, mental alienation, and epilepsy. The two latter probably depend on the same conditions that predispose to or excite the cerebral hæmorrhage.

(2) Morbus cordis, hypertrophy, fatty heart, fibrinous concretions in the heart, valvular disease, disease of the origin of the aorta and of vessels.

(3) Diseases of the respiratory organs, acting only indirectly, and leading mainly to hæmorrhage from veins or sinuses.

(4) Diseases of the digestive organs; hyperæmia of stomach, intestines, and liver (?); suppression of hæmorrhoidal flux.

(5) Diseases of the kidney, especially the small granular kidney, and this particularly when found in connection with rigid cerebral arteries and hypertrophy of the left ventricle of the heart.

(6) Diseases of the sexual organs; arrest of menstruation; venereal excess, especially in elderly people.

More rarely rheumatism, gout, and lead-poison.

In the following cases of Calmeil the effects on the brain of recent hæmorrhage, and of hæmorrhage of older standing, are well seen.

He gives a résumé of fifteen cases:

In 3 there were old cicatrices on the right side of the brain; in 3 on the left side; in 2 in the pons.

In 3 the spots of encephalitis with clot were situated on each side of the median line. In one of these cases the blood had filled the cavities of the lateral ventricles.

In 3 the spots were of the right lobe only; in 2 of the right lobe and the right lateral ventricle; in 2 of the right lobe and both lateral ventricles.

In 1 the lesion was in the left cerebral lobe only; in 1 in the left lobe and both lateral ventricles.

In 4 hæmorrhage existed in the pons, as well as in the brain.

In 3 in the cerebellum alone. In two of these cases the blood was found in the fourth ventricle.

In 2 other cases it existed at one and the same time in the cerebellum and cerebral hemispheres. In both blood existed in the cavity of the ventricle of the cerebellum.

In 5 the nervous substance of the spots was moderately softened ; in 5 much softened ; in 5 softened or disintegrated.

The spots of encephalitis with clot were examined microscopically in nine cases. They have never shown anything but fibrinous elements, liquid or scarcely coagulated globules of blood, hæmatosin, and cerebral fibres detached from each other, when the patient had only survived the attack from two to eight hours. In all these cases the clot only contained the elements of blood, and the cerebral fibres and lumps of nerve substance had been sometimes dragged along in the midst of these elements. The cerebral vessels were sometimes filled with blood in the neighbourhood of, or at a certain distance from, the morbid spots.

The recent softened spots were not constituted otherwise than those which were less soft ; they contained only more liquid fibrine and more serum.

In all the cases in which the patients had survived the extravasation of blood beyond twelve to fifteen hours, the spots contained molecular granules, and numerous granular cells, mixed with the products

above mentioned, namely, the elements that constitute the blood.

The clot, as well as the surrounding walls, contained granular cells and granules.

In cases of this kind the nerve substance was often broken into particles, and more or less disintegrated.

The calibre of the vessels of the spots was sometimes considerable.

In observation 177, where the case was one of traumatic hæmorrhage, the inflammatory spots contained a good many pus globules, independently of the products which we have generally found in patients who have lived for some days.

In eleven cases of non-recent hæmorrhage, there were found in 4, local spots in the two cerebral hemispheres only; in 2, in the cerebral hemispheres, the cerebellum, and the pons; in 2, in the cerebellum and the cerebral hemispheres; in 2, in the cerebral hemispheres and the pons; in 1, in the right cerebral lobe only.

In the cases in which the cerebral hemispheres were also injured, there were found on the right side 5 spots in one case, 5 in another, 1 in three cases; on the left side, 3 spots in one case, 2 in two, more than 20 small spots in another. When the brain and cerebellum were injured simultaneously, the number of the spots was 5 in one case, 3 in the other. When the brain, cerebellum, and pons were injured simultaneously, the number of spots was more than 5 in one case, and of an undetermined number in the other. When the brain and pons were alone affected, the number of the spots was 12 in one case, 7 in the other.

The walls of some spots were still softened in three or four cases. Some spots, in three cases, contained natural blood. In almost all the cases the principal spots were in the state of cellular cicatrix.

Hæmorrhage of the spinal cord is rare. It is more common in the upper part of the cord than in the lower. The cord is generally swollen in the region occupied by the hæmorrhage. The lesion is commonly found in the grey matter in the centre of the cord. Around the seat of hæmorrhage the cord softens, sometimes without any preceding myelitis, but often as the result of inflammatory action. Occasionally, and much more rarely than in the brain, there is found a cyst-like false membrane round the hæmorrhage. In cerebral hæmorrhage the dura mater is more liable to hæmorrhage than the pia mater, though less vascular; the cortical substance less liable to hæmorrhage than the medullary, yet more vascular; the corpus striatum more liable than the optic thalamus, which is equally vascular.

Gintrac, in 751 recorded observations of hæmorrhage of the nervous system, found—

Hæmorrhage of the meninges	.	.	172 times.
„ „ middle lobe of brain	.	.	127 „
„ „ pons and peduncles	.	.	76 „
„ „ corpora striata	.	.	72 „
„ „ cerebellum	.	.	55 „
Corpora striata and optic thalami	.	.	48 „
Ventricles (septum and plexus)	.	.	46 „
Cortical substance of the brain	.	.	45 „
Optic thalami	.	.	38 „
Posterior lobes of brain	.	.	33 „

Spinal cord	19 times.
Anterior lobes of brain	17 „
Medulla oblongata	2 „
Corpus callosum	1 „

III. VARIETIES IN THE AMOUNT OF BLOOD SUPPLY TO THE BRAIN AND SPINAL CORD.

It is scarcely necessary to do more than mention the fact of this variation. The proof of its possibility is due to the observations and experiments of men of our own day—Dr. Burrows, Dr. Symonds, and others. We meet with anæmia from various causes, and hyperæmia active and passive, general and local. Anæmia of the nervous centres is often only dependent on general anæmia. It may, however, occur when the blood itself is normal, and then it is due either to plugging of vessels, to any condition that interferes with their calibre, or to the pressure of tumour or of abscess; in all which cases the anæmia will be local, and the venous tissue will eventually pass, more or less, into a state of white softening; or it will be general over the whole surface of the brain, from deficient heart action, or over one hemisphere, from pressure on the common or the internal carotid by tumour &c. Besides the general pallor of the brain, however, and the extreme whiteness of the white matter, there is little to be seen in such cases, except where the anæmia has been not only general but of long duration. Under such circumstances, there is often some atrophy of the whole organ, with compensating increase of cerebro-spinal fluid. In one case I have met with this condition in a very extreme form, the atrophy of the whole brain

having proceeded to such an extent that the organ resembled the brain of chronic senile dementia, although the mental faculties had been preserved until within a few days of death. This atrophy is frequently seen over spots of the brain, where the interference with the blood supply has not been complete. When it occurs at the surface of the brain, there is no apparent alteration in the shape of the cerebral surface until the pia mater and the arachnoid has been stripped off, the depression caused by the atrophy being filled in by the cerebro-spinal fluid.

But cerebral anæmia owns other causes. It may be connected with general anæmia, and be caused by inanition, by dyspepsia, an indirect mode of inducing inanition, or by a drain on the system, such as menorrhagia and other exhausting discharges. The circulation through the brain is interfered with by the pressure of clot, of serous effusion, or of intra-cranial tumour. Venous stasis, such as may be met with in aggravated cases of heart disease, must impede arterial circulation. The same phenomenon may be induced by mental emotion, and notably by shock; and, lastly, the presence of an excess of the products of waste of tissue in the blood will lead to a similar condition.

Hyperæmia, that is active and general, will depend on hypertrophy of heart; and this condition is so often met with in connection with small granular kidney, that the state of the brain may be complicated with those changes in the structure of the smaller arteries that often accompany, if they do not depend on, this renal abnormality. The morbid appearances are similar,

whether the change be general or local, viz. a large number of vascular puncta on transverse section of the white matter of the brain, and in spots, especially where the hyperæmia has been more localised, a sieve-like appearance of the nervous matter, the tissue being the seat of numerous little depressions, due to the pressure exercised upon it by the dilated vessel. Added to this, the calibre of the vessels may be permanently altered, constant dilatation having given rise to a persistent enlargement of the vessel. This condition was found by Van der Kolk in the vessels of the medulla oblongata in patients who had died of epilepsy, the dilatation having probably been the consequence of the previous contraction of the arteries, the first factor in the series of epileptic phenomena. This, however, is not found in all epileptics, and, where it is, is doubtless the result of the attack rather than the cause.

Passive hyperæmia is more common, and is caused, in a word, by any obstruction to the flow of blood through the cerebral veins and sinuses. Mitral regurgitation, mitral stenosis, tumour in the neighbourhood of and pressing on the vena cava superior, the jugular, &c., disease of one of the important sinuses, any form and every degree of suffocation, even in some persons stooping or hanging the head down, may all cause general or local congestions of the brain, that can be recognised post mortem, if only the skull be opened before any other part of the body.

These appearances will vary according to the position and the degree of the congestion. The cortical

substance may be generally injected, and more or less discoloured; the corpora striata may be of a violet colour; the white substance of the brain may look as if it were sanded with little drops of blood; the injection may penetrate the pons; the vessels of the pia mater may be engorged, the internal surface of the dura mater furrowed by capillary arborisations; the sinuses may stand out with peculiar prominence, filled with venous blood; and at a stage further we may meet with extravasations either into the tissue of the brain itself or into the ventricles, or on the surface of the hemispheres; or, again, the congestion may have been the starting-point of a diffuse inflammation of the external layers of the grey matter of the convolutions, or even of a more complete encephalitis; whilst in cases somewhat rare the cerebral sinuses may give way at the spot where the blood-pressure is most intense.

The incrustation of the capillary vessels with granules and granular cells in congestion followed by inflammation of the cortical tissue will be noticed under the head of 'Inflammation.'

The spinal vessels may be distended even to an extreme extent without exercising a proportionate pressure upon the cord. The causes that have been mentioned as influencing spinal congestion are great muscular efforts, exposure to a hot temperature, especially to the sun's rays, some epidemic influence in the newly born, suppression of the evacuations, particularly of the catamenia, irritation of neighbouring organs, venereal excess, and the rigors of ague. It is certain, however, that temporary paralysis may follow spinal congestion.

Desnos describes a very peculiar case of paralysis, with rotation of the head and lateral deviation of the eyes, following a condition of intense congestion (without other lesion) of the hemispheres, optic thalami, corpora striata, cerebellum, peduncles, pons, and medulla oblongata.

Calmeil's account of the pathological anatomy of cases of intense cerebral congestion of temporary duration is of great interest.

In three cases the cranial bones were notably injected; in three the vessels of the dura mater were congested; in one case there was fibrinous coagulation in the longitudinal sinus; in one the internal surface of the dura mater was furrowed by capillary arborisations; in two the cavity of the arachnoid contained liquid blood and bloody humidity; in four the cerebral pia mater was generally congested; in three cases it was reddened by extravasated blood; in one the pia mater adhered in spots to the subjacent convolutions; in one these convolutions on the right side were swollen; in four the cortical substance of the brain was generally injected and more or less coloured by hæmotosin; in two cases it was injected, and much coloured on the right side; in one it was of a yellow colour; in seven the white substance of the brain was injected, and, as it were, sanded with little drops of blood; in two the corpora striata were of a violet colour; in four the pia mater of the cerebellum was tinted with violet by hæmotosin, or by suffusion of blood; in three, injected in its vessels; in two the injection had penetrated into the pons; in one the sinuses of the spinal cavity

were engorged with blood ; in two cases there had occurred effusions of blood under the spinal arachnoid.

Consult on these subjects—

- Hammond on 'The Nervous System.'
- Gintrac, 'Maladies de l'Appareil nerveux.'
- Rindfleisch, 'Path. Anatomy.'
- Wilks, 'Path. Anatomy.'
- Jaccoud, 'Pathologie interne.'
- Rokitansky, 'Path. Anatomy.'
- Bright's 'Med. Reports.'
- Carswell's Plates.
- Reynolds' 'System of Med.' Art: 'Apoplexy and Congestion of the Brain.'
- Calmeil, 'Maladies inflammatoires du Cerveau.'
- Beale on 'Kidney.'
- Liddell on 'Apoplexy.'
- Lionville on 'Aneurism.'
- Th. von Dusch, 'New Syd. Soc.' 'Thrombosis of Cerebral Sinuses.'
- 'Lond. Hosp. Rep.,' vol. i. p. 350.
- 'St. Barth. Hosp. Rep.,' 1872.
- 'Brit. Med. Jour.,' June 13, 1874. 'Aneurism of Brain.'
- 'Med. Record,' vol. i. 263. 'Sclerosis of Arteries.'
- 'Med. Record,' vol. i. 472. 'Congestion of Brain.'

LECTURE III.

INFLAMMATION.

DURA MATER.—Inflammation of the dura mater (pachymeningitis) is scarcely ever general, although the whole of the convexity may be involved at one time.

It may be purulent or non-purulent.

It arises from a great variety of causes. Blows or falls on the head may lead to the purulent, but much more frequently to the non-purulent variety. Syphilitic caries of the bone may lead to either form. The inflammation of the dura mater, consecutive to disease of the petrous portion of the temporal bone, is local and generally purulent, whilst not unfrequently the portion of membrane attacked becomes gangrenous. The vasa propria of the dura mater are specially liable to thrombosis. In the non-purulent form the new formation, the result of the inflammation, becomes very quickly the seat of vessels, and is composed of several layers; those nearest the dura mater being composed of compact lustrous connective tissue fibres, almost as dense as the dura mater itself; whilst the layer further removed from the dura mater is rich in cells, with small and narrow vessels, and the layer nearest the arachnoid often firmly uniting the arachnoid

to the dura mater, is remarkable for very large capillaries. It is from rupture of these vessels that the hæmorrhage spoken of in the last Lecture occurs; the blood being not unfrequently locked up in the lacunæ of false membrane poured out beneath the dura mater. From further alteration of these inflammatory products result the laminae of bone that are sometimes met with on the visceral surface of the cranial dura mater, especially in the falx cerebri and cerebelli, and still more frequently on the dura mater spinalis. So far as I know, this ossification is never preceded by the formation of cartilage. The bone, true bone as it is, is formed from fibrous tissue, this fibrous tissue being a further development of the inflammatory exudate from the vessels of the dura mater. In old cases of chronic spinal meningitis, such laminae of bone are met with, not continuous, but sporadically arranged over portions of the cord.

An inflammation of the dura mater, originally non-purulent, may become purulent by the occurrence of hæmorrhage, as diffuse suppuration of the whole neo-membrane may occur as the result of the pressure exercised upon the effused product by the hæmorrhage.

Except as a complication of tumour, or traumatic lesion, inflammation of the dura mater at the base of the skull is rare.

The dura mater may be perforated by sloughing ulceration, and in this case the condition almost invariably depends on the extension to this membrane of disease commencing in the bone in its immediate neighbourhood, or upon the irritation either of a wound

or of a foreign body pressing upon the dura mater. This foreign body is not unfrequently a portion of the cranial bones depressed on the subjacent membrane by some accident.

The spinal dura mater, however, may be secondarily attacked by the extension to it of suppurative disease of the bodies of the vertebræ.

For inflammation of the sinuses of the dura mater, see last Lecture.

Inflammation of the Arachnoid.—It is not always possible to isolate diseases of the arachnoid from abnormalities of the under-surface of the dura mater; and still less possible is it to separate morbid conditions of the arachnoid from those of the subjacent pia mater so closely blended with it.

But we do find various degrees of thickening of the arachnoid, with sometimes deposit of lymph on the upper or under surface of its visceral layer, if not of both.

The arachnoid may appear milky from two causes: either from the presence beneath it of slightly turbid fluid, such as the cerebro-spinal fluid in not quite a normal condition, or from the faintest commencement of the thickening of the membrane itself.

Thickening and opacity of the arachnoid occurs under somewhat varied conditions; thus it is not uncommonly found—

(i) In cases of small granular kidney.

(ii) In cases of delirium tremens, with epileptiform attacks. In one such case I have found chronic thickening of the arachnoid of the convex surface of

the cerebral hemispheres, with opaque spots and plates quite distinct from the Pacchionian bodies.

(iii) There may be very chronic thickening of this membrane in cases of senile dementia. In such cases, as in many other instances of chronic thickening, there is often an atheromatous condition of the cerebral vessels.

(iv) In heart disease; where we sometimes find patches of thickening of the arachnoid at the convexity.

(v) In cancer, especially of the stomach and liver; generally associated with atheroma of the cerebral vessels.

(vi) In cerebro-spinal meningitis; in which the arachnoid of the convexity is not free from disease, although the membrane of the base is chiefly attacked.

(vii) In tuberculous cases, in which thickening of this membrane is generally associated with effusion of lymph and with tubercle of the pia mater. This abnormality is, in most cases, confined to the base, but occasionally the convexity participates; and sometimes the convexity is alone attacked.

(viii) In cases of diseased spinal cord, as, for instance, where there has been tubercle of the cord; but the plates on the spinal arachnoid, which may be soft or cartilaginous or osseous, may be the result of very chronic inflammation, not associated with tubercle.

(ix) In cases of arachnitis, consequent on disease of the bone or dura mater—as ulceration, hæmorrhage, cancer, tubercle, &c.—causing pressure on the arachnoid from above downwards; or from pressure from below upwards, as from a clot in the pia mater.

(x) In cases of arachnitis occurring after severe falls, with or without fracture of the skull.

(xi) Thickening of the arachnoid is met with also where no cause can be discovered, as in the following case :—

A woman died without any cerebral symptoms. There was great thickening and toughness of the arachnoid and pia mater over the cerebellum, pons, and medulla oblongata. The condition was evidently of long standing. The floor of the fourth ventricle was in a similar condition, except that here the thickening was slightly nodular. A very large amount of fluid in the ventricles, with some softening of the third ventricle ; brain otherwise firm. Vessels healthy.

Arachnitis, with effusion of Lymph or of Pus.—This is generally a further stage of mere thickening and opacity ; certainly the effusion of inflammatory products never occurs without some decided opacity at least of the arachnoid.

The effused product is most frequently found on the under-surface of the arachnoid, and it is probable that under these circumstances the arachnoid is only secondarily affected, the pia mater being the tissue primarily inflamed.

This is especially seen to be the case in tuberculous and in cerebro-spinal meningitis. In the former case, indeed, the arachnoid, when inflamed, seems only so in direct ratio and extent with the pia mater, and the pia mater may be affected whilst the arachnoid is almost wholly or wholly free.

We sometimes, however, find the upper surface of

the visceral arachnoid coated with lymph, from irritation reflected on it from above, as from various abnormalities of the dura mater. In such circumstances the pia mater may be healthy. The upper surface of the visceral arachnoid of the convexity may be covered with lymph, whilst the subjacent pia mater is in a similar condition, as is seen sometimes after falls on the head without fracture of the skull. Occasionally also lymph is found on the upper surface of this membrane under circumstances which can only be considered due to inflammation. This was seen in the case of a woman who died with right hemiplegia and softening of the brain; the two cerebral hemispheres were united together over the corpus callosum by soft adhesions on the arachnoid.

I published a case in 'St. George's Hospital Reports,' Vol. 4, of a young man who had died of an attack of hæmorrhage from rupture of the right middle meningeal artery; the dura mater all over the convex surface of the hemispheres was somewhat adherent to the subjacent arachnoid, whilst the arachnoid was thickened and yellow all over. This patient had been under a surgeon's care a year before with great pain over the whole of the upper part of the head, and had been treated with entire success with iodide of potassium.

Although it is difficult to separate the abnormalities of the pia mater from those of the arachnoid, yet it is generally easy to speak with certainty of these abnormalities as having their origin in the pia mater.

1. Meningitis of the convexity is much less often accompanied with softening of the central portion of

the brain than meningitis of the base. Hyperæmia of the vessels is seen first, and there generally results an effusion of pus or lymph in the sub-arachnoid spaces. This may be partial—over a spot, for instance, no larger than half-a-crown—or general over the whole convexity. Sometimes, though rarely, both convexity and base are attacked in the same manner. The pia mater may be affected very intensely, and the arachnoid hardly at all. The congested vessels and the effused products are seen occupying the sulci between the convolutions. As a general rule, meningitis of the convexity is not associated with tubercle, whilst meningitis of the base is tuberculous; but to this there are many exceptions. Thus in one case ‘a good deal of lymph, mixed with granules of tubercle, was found on the convex surface of the left hemisphere. A similar condition over base. The lymph seemed to dip in between the convolutions, following the course of the pia mater.’

Again, in another case, ‘the arachnoid and pia mater, at the upper parts of the hemispheres, contained much clear fluid, but were natural; between the hemispheres of the brain, in the longitudinal fissure, were many miliary tubercles; and at the lower part of this fissure, the opposed hemispheres were adherent to each other by means of a mass of scrofulous matter, equal in size to a hazel nut. A small portion of a similar growth was found at the upper part of the cerebellum, connected with the arachnoid.’

As an instance of non-tuberculous meningitis of the base :

‘Woman, aged 18. On the under-surface of the arachnoid, along the course of the blood-vessels,

much soft material existed at the upper and lateral parts of the cerebral hemispheres; and at the base of the brain, at a point chiefly corresponding to the base of the third ventricle, and the pons, the membranes were adherent to the cerebral tissues by means of a thick deposit of the same soft material.'

If the disease is of slight intensity, and over a region of very limited extent, the effused products may be reabsorbed. The only abnormality attending this condition may be a slight atrophy of the cerebral convolutions immediately subjacent to the spot of thickened membranes, and a corresponding increase of cerebro-spinal fluid at the same spot.

It will be seen in a future lecture how a thickened state of the pia mater is constantly found in cases of general paralysis of the insane.

Whether meningitis ever occurs independently of syphilis, rheumatism, alcoholic poisoning, tubercle, anæmia, or mechanical irritations is at least an open question. Dr. Bright, however, gives a beautiful plate of puriform fluid beneath the arachnoid. If cerebro-spinal meningitis is excluded, the only case I have known was that of a young lady, whose mother was rheumatic, although she herself had never suffered from rheumatism affecting the joints or the heart. But it certainly occurs from irritation from above downwards, and that over a space corresponding to the extent of the irritating lesion. Thus it is not unusual to find thickening of this membrane over surfaces subjacent to diseased cranial bones, ulcerated dura mater, &c.; or pressed upon by exostoses or other tumours from

above: and the St. Bartholomew's Museum contains a specimen of the pia mater opaque and indurated over the convexity of the brain and between the convolutions, in a case of hæmorrhage outside the dura mater. This non-tuberculous meningitis may be chronic. Plate 1 is taken from a specimen of this kind, from the collection of Van der Kolk in the Oxford Museum.

2. *Cerebro-spinal Meningitis*.—Sometimes sporadic, generally epidemic. The post-mortem appearances are not always the same, even where death occurs at precisely the same stage of the disease.

As an instance of the sporadic form—

‘Man, aged 28. Dura mater healthy. On removing the dura mater a yellow spot, the size of half-a-crown, was seen beneath the arachnoid on the anterior convolutions on the left side, close to the central division. This was composed of lymph effused between the arachnoid and the pia mater. The whole of the convex arachnoid was milky in appearance. At the base of the brain the whole of the region comprehended in the circle of Willis was covered with a compact thick yellow false membrane, in which the nerves were imbedded. In a very slight degree it existed in the fissures of Sylvius, but it became intense at the infundibulum, covered the pons, the medulla oblongata, and part of the cerebellum, and ran up between the lobes of the cerebellum. Many puncta on section of the brain. Much turbid fluid in the ventricles, and the lateral, the third, and the fourth were lined in their lower part by a similar yellow coating of lymph. The septum, and much of the central white matter around the ventricles were creamy.



Choroid plexus very tortuous, and covered with yellow lymph. The spinal cord was covered on its cervical portions with yellow lymph.'

The most usual appearances are great vascularity of the dura mater, both of the brain and cord, and sometimes extravasation of blood outside the spinal dura mater. The *diplœ* of the bones is often highly injected. Great congestion of the cerebral sinuses. The pia mater and the arachnoid are injected, sometimes more or less thickened; the layers of the arachnoid may be adherent by means of false membrane. Sometimes there is fluid in the cavity of the arachnoid, especially in the spinal canal. Exudation is very frequently found beneath the arachnoid: very often this is purulent, and occupies very various seats. It may be situated over the whole convex surface of the brain, and at the base of the brain, and over a considerable portion of the spinal cord: or it may occupy a very small portion of the convexity of the brain, or more frequently it is found over a great part of one surface of the cord, especially the posterior. In the lower portion of the cord the whole organ may be surrounded by pus. There is often a turbid, serous, or purulent exudation in the lateral ventricles of the brain, and the vessels of the choroid plexus are much engorged.

The condition of the brain and spinal cord has seemed to vary in different epidemics. Sometimes the organs are healthy, or nearly so; in other cases the external cortical layer is found adherent to the pia mater, and is detached with it. In others, again, there is simply hyperæmia, or more or less induration; but

softening is more common, varying much in degree, but not unusually very general.

In Dr. Collins' cases, microscopical examination of the exudation showed that it consisted of granular débris, imbedded in which were numerous small granular cells, chiefly circular in form, measuring about $\frac{1}{4000}$ of an inch in diameter, very similar in character to pus corpuscles, save that they were much smaller in size. He says, too, that 'Mayne long ago drew attention to the fact, that the nervous matter seldom suffered, and, when involved, was only so accidentally, owing to the propagation of its disease from the membranous investments. We can fully bear out this testimony in the cases we have observed, both as regards the brain and spinal marrow. The cords we have examined with care, but in only one of them could we find any evidence of disintegration under the microscope. Specimens, however, have been submitted to the Pathological Society of Dublin by Drs. B. M'Dowel and Hayden, in which the marks of localised softenings of the spinal cord were present. Softening of the substance of the encephalon, though also uncommon, appears to be more frequent than the corresponding condition of the cord. Allusion is here made only to cases of white softening, but Klebs, on two or three occasions, in rapid cases of cerebro-spinal meningitis, has found foci of softening, varying in shade from straw-colour to red in the centrum ovale, and in each of these cases he has found indications of recent endocarditis.'

3. *Acute Basilar Meningitis*.—This is said by Gintrac to be sometimes non-tuberculous. It is certain that

meningitis of the base, and of the base only, exists when it is impossible to detect the granulations of tubercle with the naked eye. This, however, is no proof of its non-tuberculous origin.

For practical purposes it may be considered either a syphilitic or a tuberculous meningitis; and when tuberculous, it is generally associated with the presence of tubercle elsewhere, so generally that the rule is almost without exception at all ages beyond early childhood, though in the first five years of life the exceptions are slightly more numerous.

With reference to the situation of tubercle, Dr. Green's account is very clear: 'The sheaths of the arteries, especially in the fissures of Sylvius, are the preferred seats of development. The tubercles are placed as lateral projections, singly or united into small groups upon the larger branches, while upon the more minute branches and the almost capillary vessels they present themselves as spindle-shaped varicosities, which occupy the whole circumference of the vessels. They injure the lumen of the vessel, at first by pushing into the tunica media and intima, and then by perforating them. This interference with the calibre of the vessels may give rise to congestion and hæmorrhage; and if active hyperæmia supervenes, basilar meningitis is the result.'

When this condition of meningitis is set up, the whole of the base of the brain is often covered with effused products, especially with a softish lymph or pus, matting together all the nerves as they emerge.

Occasionally some portion of the convexity is implicated as well, and it is not unusual for the convex

surface of the cerebellum to be affected. There is dryness of the arachnoid of the convexity. A venous congestion of the pia mater there; sometimes a little lymph in the sulci, and occasionally, on removing the pia mater, a small portion of the external layer of the convolutions is removed adherent to it. Tubercle is generally found not only along the fissures of Sylvius, but coating a considerable space over the infundibulum and the neighbouring organs.

This abnormality of the pia mater is almost universally associated with more or less distension of the lateral ventricles with slightly turbid fluid, and with softening of the septum, and, indeed, of all the white matter forming the roof and walls of the ventricles.

The third, fourth, and fifth ventricles may be similarly distended and their walls softened. The ependyma of the ventricles is thickened and raised into small granular elevations by collection of cells. Sometimes small tubercles are seen on the velum interpositum.

Capillary hæmorrhages of the cortical substance are not unfrequently coincident with basilar meningitis.

Tubercles are very seldom found in the plexus choroides, but they are met with in situations in which there is no pia mater, as on the walls of the ventricles, on the internal surface of the dura mater, the parietal layer of the arachnoid, or the free surface of the visceral arachnoid.

4. *Hydrocephalus*.—This may be acute or chronic. I cannot agree with authors who draw a marked distinction between acute hydrocephalus and the condition of the ventricles in tuberculous meningitis. It is said by some that acute hydrocephalus is a flux, and should

show little or no trace of inflammation; that it is best described by the old term, 'Water-stroke;' that it is a flux caused by extreme fulness of vessels, especially if the blood is spanæmic, as it may be in serofula, tubercle, cachexia, albuminuria, &c.; and that this is the connection between these conditions and acute hydrocephalus. This latter abnormality, in most cases at least, seems not to be due to any impediment in the course of the *venæ galeni*, the passage of the blood being generally found free from all obstacle. It is really due to the inflammation of the *ependyma*, a condition that may take place quite by itself, without any implication of other parts of the brain or membranes, except indeed the portions of the brain immediately subjacent to the *ependyma* itself. It is not absolutely necessary that it should be associated with tuberculous meningitis, but it very often is so, and in almost all cases seems to depend upon the same causes. In a word, acute hydrocephalus is generally tuberculous meningitis, with the lining membrane of the ventricles more implicated than is usually the case in ordinary meningitis of the base. This inflammation of the *ependyma*, with effusion more moderate than in acute hydrocephalus, is not an unusual appearance in several conditions of brain disease. It may be found in chronic hydrocephalus, in general paralysis of the insane, in chronic mania, and sometimes also in melancholia.

An instance of this was seen in a man who died with pulmonary abscesses.

Skull cap very thick. Dura mater very adherent all along the superior longitudinal sinus. This sinus

was empty. More than four ounces and a half of fluid was collected from beneath the arachnoid. This membrane was milky and slightly thickened everywhere. Cerebral hemispheres tolerably firm, a little flattened at the anterior portion. More than three ounces of fluid in the lateral ventricles. Their cavities enlarged, and walls almost diffuent. The third ventricle was opened out by the pressure of fluid, and the soft commissure destroyed. The lining membrane of the lateral, third, and fourth ventricles was much thickened and very granular, being quite rough to the touch. It was impossible to detach it from the subjacent brains. This granular condition was composed of granules, small cells, and a few exudation corpuscles. The membrane surrounding the pituitary gland was thick and white. Cerebellum healthy. Pons and medulla oblongata rather soft.

5. This seems perhaps the most convenient place to say something about chronic hydrocephalus, although inflammation plays a less important part in its causation than in the case of the acute condition. Still, a low form of inflammation attacking the arachnoid lining of the ventricles, during foetal life and in earliest infancy, sometimes even later in life, is more often the cause of this peculiar abnormality than has been recognised by some authors. Dr. Bright's plates of 'Cardinal' are well worth study on this subject.

I find few accounts of post-mortem appearances that seem to justify the idea of external hydrocephalus. Rindfleisch's account of the causation of the disease appears to be wholly theoretical. He says: 'The seat

of this disease is the region of the cerebral veins of the vertex along the longitudinal sinus; the first result being elongation of the veins and dilatation; second, œdema of the pia mater and effusion of serous fluid. The gyri are pressed asunder, the sulci dilated. Decrease of thickness in the hemispheres. Milky clouding of the pia mater due to infiltration of colourless cells, and even afterwards a gradual fibrous transformation.'

All this is little more than a description of what occurs in atrophy of the brain or defective development, in which the cerebro-spinal fluid takes the place of the atrophied convolutions; and the membranes over the convexity appear at first sight like a bladder partly filled with water. Those cases of reputed external hydrocephalus, and of recovery after tapping the head, were either instances of this atrophy of the brain or of internal hydrocephalus, in which the pressure of fluid from within had caused absorption of a large amount of the brain substance, and the trocar had passed not only through the bone and membranes, but also through the thin layers of cerebral substance surrounding the mass of fluid within, or some sudden accident had caused the rupture of this thin layer of brain substance, and the fluid which originated internally had escaped through the rent to an external situation.

It seems on the whole most in accordance with experience to say that external hydrocephalus has no existence, except in those rare cases in which fluid in the cavity of the arachnoid is the result of hæmorrhage into the arachnoid sac.

Chronic hydrocephalus of the ventricles is sometimes a result of the acute disease, or, at any rate, is a consequence of inflammatory action. Dr. West, in his sixth edition of his work on diseases of infancy and childhood, says: ‘I may further add that each year leads me to estimate more highly the share of inflammation of the lining membrane of the ventricles in the production of chronic hydrocephalus.’

In many cases, however, it is a simple flux depending on some impediment to the flow of blood through the *venæ galeni*. It has been seen as the result of closure of the fourth ventricle by a thickened arachnoid; also as a consequence of disease of the choroid plexus, which is sometimes tuberculous.

When not tuberculous, the surface of the choroid plexus is found beset with numerous papillæ, consisting of a thick mantle of epithelial cells, and a central part made up of the lumina of vessels.

A cystic condition of the choroid plexus may be found without hydrocephalus resulting from it; but a very intense form of this disease would lead to a very considerable effusion.

Plate 2 represents a very unusual condition of cysts of the choroid plexus from the Oxford Museum.

It is easy to understand how the reflux of the venous circulation from the brain would be interfered with by a tumour attached to the choroid plexus, such as is seen in specimen 811, one of Van der Kolk's, in the same museum: ‘in the left ventricle is a large white tumour of moderate consistence, resembling brain substance in appearance, but not springing from the



CYSTS OF CHOROID PLEXUS.

brain, only loosely adherent to the choroid plexus. There was no mania nor paralysis.'

The anatomical appearances of chronic hydrocephalus vary according to the amount of fluid, and to some extent, also, according to the period of life at which the disease originated.

These appearances are, distension of the ventricles, compression of their walls, and especially of the floor of the ventricles, a very patent condition of the channels which lead from one ventricle to another, thinning or even unfolding of the superjacent convolutions, a thickened ependyma, if the disease has had an inflammatory origin. The state of the bones varies considerably; either the sutures have never united, and a large portion of the calvarium is not ossified; or the union is less firm than normal, with the interposition of many ossa triquetra; or the bones are much thinned; or, lastly, the bones are found either thicker than usual, or of the normal density.

The pressure of a tumour may frequently, by irritation, set up a certain amount of meningitis in its neighbourhood. This, being generally very localised, is only of importance if it implicates any nerves. As a matter of fact, meningitis so caused does sometimes thus implicate the optic nerves, and the low form of inflammation spreading to them may lead to true optic neuritis, or, by causing increase of connective tissue growth in the nerve, induce atrophy.

6. *Spinal Meningitis*.—Very often spinal meningitis occurs coincidently with cerebral, as in sporadic and epidemic cerebro-spinal meningitis, and occasionally also in the tuberculous form.

The post-mortem appearances vary somewhat according to the cause of the disease. It may be that the disease has been set up by lesion of the bones or of the ligaments; and we find corresponding alterations in these organs, or the dura mater is thickened and adherent to the bone, or gangrenous. The arachnoid is less often primarily attacked than the pia mater, but is sometimes the seat of opacities, and in chronic cases even of cartilaginous or bony plates. The pia mater is the most usual seat of inflammation. The vessels are turgid, and there is effusion of clear, or flocculent, or sanious, or purulent fluid. The spinal cord may be somewhat compressed by the fluid and its vessels empty; or it may be injected, or softened, or indurated; and, lastly, it may in no way participate in the disease of the membranes.

It is not very uncommon to find thickening of the spinal meninges in cases of fatal tetanus.

Encephalitis.—This term will include inflammation of any or all the contents of the cranium, except the meninges and the nerves. Meningitis, however, is a frequent complication.

It may be general, or at any rate diffused in many spots over the various organs contained in the cranium, or it may be confined to one region, or to one organ. The cortical substance alone, or the grey and white matter in any one lobe, may be affected. If, however, one lobe is attacked, the opposite corresponding one will most probably suffer in unison, unless the inflammation is from a local traumatic cause. The great centres at the base of the brain, the corpora striata

and optic thalami, either singly or together, or the crura cerebri, the cerebellum, the floor of the fourth ventricle, and the pons, may be the seat of this abnormality. The whole of the medulla oblongata has been said to have been so affected; but this condition is extremely rare.

The post-mortem appearances vary exceedingly, according to the conditions under which the disease is set up. They are partly recognisable by the naked eye, and partly to be realised by high magnifying powers. Of the first, the appearances vary according to the stage of the disease at which the examination is made, and also according to the chronicity of the disease itself. Thus the lesions are:—

(i) Injection of the cortical substance, with or without a certain friability of this substance, and most commonly of the external layer.

(ii) Or injection of both white and grey matter; this condition of the white matter being evidenced by the numerous bloody puncta on transverse section.

(iii) Spots or regions of induration, especially of the grey matter.

(iv) Spots or regions of red softening.

(v) The formation of pus, either as one large abscess, or in multiple abscesses. These abscesses may be bounded by a cyst-wall, or may merge gradually into regions of softening; or they may cause bands of secondary degeneration.

(vi) A gangrenous appearance, generally very localised.

In connection with the fourth variety of this series

is a form described by Dr. Moxon and others, and named by him 'Congenital Encephalitis.' Its characters are softened patches containing granule cells (which represent fattily degenerated neuroglia cells), spindle-shaped clear bodies, not provided with nucleus or nucleolus, and caused, according to Virchow, by swelling and severance of the axis cylinders of the nerve tubercles. The proper tissue of the brain in these cases is almost utterly destroyed by hyperplasia of the neuroglia.

Taking these coarser varieties first, we find under the head of injection some differences, according as the disease is acute or chronic. In the acute form the calibre of the capillaries is increased: there is sometimes an increase of the sub-arachnoid fluid both over the convex surface of the hemispheres and in the ventricles.

The vessels of the choroid plexus are often large; the white matter of the brain seems increased in density, probably from pressure. This pressure is explained by the appearance of the vessels in the more chronic form. There is often a tortuous condition of the minute vessels; sometimes they are aneurismal, occasionally also they have given way, and small granules of hæmatine are found in the immediate neighbourhood of vessels; whilst the hemispheres show on transverse section a cribriform appearance, from the presence of numerous little holes, probably caused by the pressure of the small vessels, enlarged during life and contracted after death.

Of the spots of induration, an occasional form of

degeneration that sometimes owns an inflammatory origin, something will be said by-and-by, under the head of Sclerosis.

Red softening gathers its colour from minute hæmorrhages; but non-inflammatory softening, depending only upon obstructed circulation, may be red from hæmorrhages or from capillary congestion. The softened mass is composed of pus, disconnected fragments of nerve fibres, granular cells, and pigmentary ganglion cells, but all the appearances hitherto supposed to be characteristic of inflammatory softening have been found in softening manifestly non-inflammatory; so that I confess myself unable to determine, by post-mortem appearances alone, whether a patch of softening has depended on an inflammatory origin or no.

When abscess is formed, the pus comes partly probably from proliferation of the neuroglia cells, but mainly from the emigration of white cells from the vessels.

Beyond the dépôt of pus proper we meet with a zone of œdematous saturation, by which both nucleus and cell swell up. Calcification and excessive pigmentary infiltration may occur. Cerebral abscess is generally encapsulated by a thin compact connective tissue capsule. This capsule is formed from the neuroglia, and is composed from within the abscess outwards. It is composed of: (1) A layer of fatty degenerated cells; (2) A layer of germinal tissue, which by its irregular thickness causes wave-like irregularities of the surface. This merges into (3) very

loose spindle-shaped tissue, which is stratified distinctly parallel to the surface ; (4) fatty granular cells, making a zone of yellow softening at the boundary of the abscess membrane towards the brain. (Rindfleisch.)

The following case is an example of cerebral abscess :—

‘ Man, 38. Dura mater healthy, except at a spot at the upper and posterior portion of the middle right cerebral lobe, where it was very adherent to the subjacent tissues. The longitudinal sinus close to this spot was full of a light-coloured clot. At this spot, and extending forwards, and laterally over a space about two inches square, the convolutions were flattened smooth and shining, and darkish with the colour of an old bruise. On removing the arachnoid and pia mater, about the centre of this patch the brain substance gave way, and a little healthy pus escaped. On enlarging the opening a cavity was shown, large enough to contain a small orange, and full of pus. The roof of this cavity was found mainly of grey matter about a quarter of an inch thick, except in the one spot where the brain substance had softened, and allowed the pus to reach the convex surface of the hemisphere. This cavity was lined with a firm tense roughened false membrane, about two or three lines thick, forming a kind of cyst. The abscess lay wholly in the white substance of the hemisphere, and did not reach the ventricle at all. Cerebral vessels healthy.’

It is quite possible to find in different spots in the same individual depôts of pus encysted in the manner just described ; pus encircled with cerebral tissue, soft

or indurated, or much injected, but not forming a regular cyst-wall, and mere regions of softening, of which the centre is softer than the circumference, the latter gradually losing itself in the healthy parts.

Of multiple abscesses, the number is generally in inverse ratio to their bulk.

The white and grey matter are almost equally liable to inflammation. The middle lobe is a frequent seat; the anterior and posterior less so. The corpora striata, optic thalami, and ventricles are often the seat of inflammation; the cerebellum and pons are less subject to it; but many parts may be affected simultaneously.

The stages, then, of encephalitis are, according to Rindfleisch: (1) Hyperæmia. (2) Infarction. (3) Œdema, and in some cases hæmorrhage. (4) Proliferation of the elements of the brain tissue, cellules, and nuclei. (5) Hypertrophy. (6) In some cases, especially in chronic cases, Induration, generally not occupying the whole seat of inflammation, but accompanying softening either at the centre or at the circumference. (7) Softening; when inflammatory, accompanied by a small infiltration of blood. The softened region contains inflammatory granules, which also, however, exist sometimes in non-inflammatory softening (as also do the small hæmorrhages). (8) Suppuration. The cyst round the abscess may begin to form from the 13th to the 24th day. (9) Rarely false membrane. (10) A condition of fœtid suppuration or softening, resembling gangrene. (11) More or less atrophy, the result of inflammation. Many instances

of so-called acute abscess have really been examples of local softening, due merely to intercepted blood supply.

It is very remarkable how seldom we meet with abscess of the brain that may be called idiopathic. Thus of 76 cases collected in Reynolds' 'System of Medicine,' 18 were secondary to suppuration elsewhere, 17 were due to direct injury of the head, 27 to disease of the internal ear or of the petrous portion of the temporal bone, 3 to various diseases of the nose, 1 to syphilitic disease of the bones of the head, 1 to carcinoma of the roof of the orbit; in 8 the cause was unknown, and in only 1 case could the abscess with any probability be termed idiopathic.

I add the following cases, as in each the cerebellum was affected—a rare condition :—

Man, 23. Numerous abscesses, of a size varying from a pin's head, at various parts of the brain. In the right hemisphere, almost half an inch from the surface, were two small abscesses, each about the size of a filbert. In the left hemisphere, about its centre, was a small abscess, surrounded by a number of small collections of pus; there were two others, one in the back part and another in the front part. Much purulent lymph in the left lateral ventricle, at the back part, and filling its descending course. Septum lucidum soft. Separate collections of pus in the left hippocampus major and left optic thalamus. A small abscess in the centre of the cerebellum. Each of these collections of pus was surrounded by a zone of congested vessels, and appeared of recent formation. There were also many abscesses in both lungs.

Man, 23. Occasional nystagmus. Left pupil larger than the right. Left arm weaker than the other, and was tremulous at times. No other paralysis. Intense intermittent pain over the whole head. No vomiting. No loss of co-ordination. Sight very defective. Optic neuritis of left eye.

P. M. Ex. Brain quite healthy, except that the lateral and third ventricles were very much distended with clear fluid. A very little softening of the septum. In the centre of the left hemisphere of the cerebellum was an abscess, lined with thick false membrane, and containing nearly one ounce of pus. Sinuses healthy. Left internal ear healthy. Other organs healthy. This seemed to be a case of idiopathic abscess.

There are other conditions, not recognisable without magnifying powers :

1. Changes in the neuroglia.

General sclerosis—rare.

Disseminated sclerosis.

Miliary sclerosis.

2. Changes in the vessels.

Microscopic aneurism, or rupture.

Dilatation of vessels.

Results of exudation in the tunica adventitia.

Masses of hæmatoidine, or of pigment, deposited outside the vessels.

3. Pigmentation of cells.

Sometimes large cells are found in the middle layer of the cortex, with many large branches.

Small granular cells are often rapidly formed.

Of the fifteen cases given by Calmeil under the head of acute cerebral softening, or local acute encephalitis without clots of blood, he found in one fibrine in the sinuses of the dura mater; in one this membrane was bathed in purulent liquid, and it was also perforated at one point.

In five there were recent spots of encephalitis on the right and left sides, in six on the left only, in three on the right only.

In three there were cellular cicatrices in the right lobe of the brain, in one in the left lobe.

In two the right hemisphere of the cerebellum was the seat of an acute inflammatory spot.

In four the principal recent inflammatory spots were still in a state of red hepatization. In seven they were in a state of softening, with disintegration of the nervous substance. In four they were in a state of disintegration of the nervous substance, with a mixture of a liquid that resembled pus.

In four the spots of acute local encephalitis without clot were studied microscopically. Of these, in one they were still in the state of red hepatization; the diseased regions were reddened by the widening of the capillaries, and by the presence of extravasated globules of blood; the cerebral fibres were not yet disintegrated; already small granular cells had begun to be formed in the inflamed parts.

In three the nervous substance of the diseased seats was disintegrated, and more or less reduced to fragments; it was soaked in plasma, mixed with a considerable number of great cells collected together,

and molecular granules; sometimes in the preparation there were seen rare globules of pus scattered. The vessels and their principal branches were constantly very apparent.

Myelitis.—Opinions are divided as to the nature of the change in so-called myelitis. Some observers affirm that what is called myelitis is only a form of meningitis, an inflammation of the connective tissue supporting the cord; that inflammation of the spinal cord is of an interstitial character, leading to the degeneration and disintegration of the nerve structure instead of the production of epithelial elements and pus.

Without doubt, also, the breaking up of the softening surrounding a clot in the cord, or a scrofulous mass, has not unfrequently been mistaken for abscess of the spinal marrow.

Still, inflammation, with true inflammatory products, may be found in the cord, involving not the neuroglia only, but the other component parts of the white and grey matter; but under all circumstances the neuroglia is probably the first and always the main structure affected.

Inflammation, according to Hammond, Jaccoud, and other observers, may attack the grey and white matter (in which case it becomes impossible to distinguish the two substances from each other) or one only. When it affects the white matter, the membranes of the affected portions are congested, thickened, opaque in patches, and adherent to the cord. The cord is softened to a variable depth, and detached in pieces

with the membranes on their removal. The softened portion is in the early stage rose-coloured and studded with red points, marking the situation of the enlarged blood-vessels. As the disease advances, the colour deepens to a reddish-brown, then becoming lighter, passing through several shades of yellow, until at last all colour is lost. Similar changes are seen in the grey matter.

Microscopically we find congestion and increase of the connective tissue of the cord. The evidences of this hypertrophy are seen in the increase of fusiform cells, and in the production of multi-nuclear cells and free nuclei. These formations take place at the expense of the proper nervous tissue of the cord, the anatomical elements of which undergo atrophy and fatty degeneration. The nervous tubules are thus often disintegrated, and their contents disseminated through the extraneous tissue. The axis cylinders are entirely surrounded by oil globules, or altogether broken up. Should suppuration occur, the elements of pus are observed among those already described, and take their place to a considerable extent. As acute myelitis becomes chronic, the centre of inflammation undergoes other changes, still characteristic of hypertrophy of the neuroglia, at the expense of the proper nervous tissue. Sclerosis is the result.

Occasionally, however, the softening persists, and becomes the permanent structural condition of the diseased portion of the cord. These conditions of softening and induration of the cord, like similar changes in the brain, seem to mark the acuteness or chronicity of the inflammatory action.



Michaud's plates give an excellent idea of the extent of degeneration and distortion the cord may undergo from inflammation attacking either its substance or its meninges. They also show very beautifully the gradual passage of inflammation into sclerosis, or rather the mode of origin of sclerosis from inflammation.

The condition of softening will very generally affect the grey matter first, probably from its greater richness in blood-vessels; it may be found only in one spot, or in a few isolated spots; but it has been met with along the whole length of the cord.

Hæmorrhage often occurs in the softened portions, and may even be of considerable extent. Such cases must be distinguished from inflammatory softening round hæmorrhages, the hæmorrhage being the primary lesion.

Myelitis may co-exist with chronic cerebral softening from encephalitis. Calmeil mentions three such cases, in one of which the cord was thin and indurated, in one softened and disintegrated, in one of a violet colour.

Plate 3 is an example of myelitis following fracture of the bodies of some of the vertebræ and consequent hæmorrhage. Plate 4 shows how completely the nervous substance of the spinal cord can be destroyed by myelitis. The case was as follows:—

Fanny O——, aged 37, married. Has been ill for the last fourteen days. Was first taken with numbness in the left leg and motor paralysis; the right leg became gradually affected in the same way

a few days later. She lost her sight suddenly fourteen days ago. Was then constipated, and had difficulty in passing water. On admission is almost wholly blind; she can just distinguish the form of a person near her, but not the features. Is quite paraplegic, being unable to move the leg in the least degree. Has lost sensation to a great extent over the whole region of the motor paralysis. No power over the sphincters. Urine dribbles away, and yet much is retained, and has to be drawn off by the catheter. No paralysis of the upper limbs, or of face, or of the muscles of the eyeball. Retinal vessels very small, and discs cloudy. Is very hungry and very thirsty. Has a large deep bed-sore on each buttock, and her feet are blistered in many places from having been burnt by hot bottles. Complains of pains in the arms. Abdomen tympanitic. She feels cold in the legs, though anæsthetic to touch. Has been subject to pain in the head ever since her marriage. Four days after admission she was able to see much better. At the end of three weeks she was beginning to move the right leg, but she died in about a month from exhaustion from the bed-sores.

P. M. Ex. Brain healthy. Spinal cord, at a point corresponding to the last dorsal and first lumbar vertebræ, was much disorganised, being a dark brown over much of its thickness. A similar condition, though of less degree, existed about the middle of the dorsal region. Plate 4 shows very little remaining besides the vessels, with some thickening of connective tissue, and a somewhat gelatinous appearance of what remained of the columns of the cord.

Dr. Von Rabenau describes three cases of chronic mental derangement, in all of which there were peculiar alterations in the posterior columns of the cord, due to chronic myelitis.

Consult—

- Hammond, 'Diseases of the Nervous System.'
 Calmeil, 'Maladies inflammatoires du Cerveau.'
 Reynolds' 'System of Medicine.'
 Rindfleisch, 'Path. Histology.'
 Bright's 'Med. Reports.'
 Green, 'Path. Anatomy.'
 Gintrac, 'Maladies de l'appareil Nerveux.'
 Wilks, 'Path. Anatomy.'
 Rokitansky, 'Path. Anatomy.'
 'Med. Times.' 1870. Dr. Moxon.
 'Jour. of Mental Science.' July 1873. Dr. Jastrowitz.
 'West Riding Asylum Rep.,' i. 218. 'Arachnoid Cysts.'
 'St. Barth. Hosp. Rep.' 1872. Mr. Kesteven.
 Dr. Collins' 'Report on Epidemic Cerebro-Spinal Meningitis.'
 'Privy Council Rep. on Cerebro-Spinal Meningitis.' Drs. Whitley and Sanderson.
 West, 'Dis. of Infancy and Childhood.' 6th edit. 'Ch. Hydrocephalus.'
 'St. George's Hosp. Rep.,' vol. iv. 'Tuberculous Meningitis.'
 Dr. E. L. Fox.
 'Med. Record,' i. 296. 'Myelitis in the Insane.'
 'Michaud, 'Sur la Méningite et la Myélite,' &c.

LECTURE IV.

DEGENERATIONS.

DEGENERATIONS include atrophy, softening, and the various forms of sclerosis.

The distinctions between these varieties are arbitrary, and can only be defended on the ground of convenience. Thus atrophy may depend upon, or be intermingled with, softening, or, again, with sclerosis. It may be due to inflammation, as also may at least one variety of softening, and it is an open question whether all forms of sclerosis do not own a similar inflammatory origin.

Atrophy may be met with under several forms. Thus we find distinct losses of substance, forming cavities, either at the circumference of the organ or in its interior; or there may be a general wasting of the whole contents of the encephalon or the spinal canal, the space formerly occupied by the nervous substance being filled with cerebro-spinal fluid; or more commonly there is diminution in the bulk of one organ or a small part of an organ, of a hemisphere of the brain or cerebellum, or of one lobe of either, or one half of the pons, of one corpus olivare, &c.

It may be a congenital condition, or one acquired

as a sequence to an accident, or to some disease, especially anything that slowly interferes with the nutrition of the organ. Of the first variety, in which there is a distinct loss of substance, it is most usual to find one spot in the brain, but in some few cases there are several. The cavity is generally lined with a thin membrane of great vascularity; the sides of the cavity are somewhat indurated; the contents may be pultaceous, or only a turbid fluid. It is probable that this form is strictly a sequence of inflammation.

Far more frequently we meet with a gradual loss of bulk in some organ or part of an organ, the general relations and shape of the organ being retained.

In the brain the volume and weight of the organ are diminished. The whole brain, or at least all the convolutions of the convex surface of the hemispheres, may be found sunk, and their place taken by a considerable amount of fluid. This is common enough in old age, with feebleness of mental effort, in chronic mania, and dementia. I have met with it, however, once in the brain of a very intellectual person, who retained not only consciousness, but brightness of mental power to the last; in this case it was associated with sclerotic lesions of the cord. Unless it depends on senile conditions, it is frequently associated with various deformities of the cranial bones, thickening of the meninges, and patches of meningeal ossification, with fluid in the ventricles, or, if the atrophy is unilateral, with fluid in the ventricle of the affected hemisphere, with thickened ependyma, and indurations of the ventricular wall. All these concomitant lesions

point to an inflammatory origin for the atrophy ; the process in order of sequence being inflammation, then softening with fatty degeneration, then partial absorption, and so atrophy. Sometimes the brain is diminished in thickness, sometimes in length. The cerebellum may be atrophied in the half opposed to that of the brain ; thus atrophy of the right cerebellum may coincide with atrophy of the left cerebrum.

If the lesion have an inflammatory origin, we find traces of fatty degeneration of all the tissues of the vessels, the nerve-tubes, and even the cells, with débris of broken-up nerve-cells. Sometimes the neuroglia itself has softened down in the same manner ; at other times, when the inflammatory action has been of less intensity and more chronic, this tissue will have been the seat of proliferation, and some sclerosis is the result. Amyloid bodies will also be found most generally.

If the lesion have no inflammatory origin, we meet only with a shrunken condition of the nerve-tissue, especially of the calibre of the nerve-tubes. The cells seem smaller than usual, or, if the disease be unilateral, the cells are proportionately smaller than those of the corresponding portion of the brain on the unaffected side. The size of the arteries is often somewhat decreased, or the lumen both of arteries and capillaries may be found encroached upon by atheroma. In this case the border line between atrophy and softening is a very fine one, and the two lesions are conditioned very much by the gradual manner in which the disease of the vessels occurs ; any rapid interference with their calibre leading probably to softening, more gradual interruption to a free circulation causing atrophy.

A marked case of atrophy of the brain, with absence of the corpus callosum, is reported by Dr. Palmerini, and quoted in the *Medical Record*, December 31, 1873. The patient, a girl, was born of healthy parents; but when a year old showed signs of mental weakness, and grew up in this state till her sixteenth year, when she was received into the asylum at Siena. Her skull was small and symmetrical, her forehead small, the teeth long and large; her aspect was imbecile, and her growth stunted. All the mental faculties were very weak; she did nothing except by imitation, or in obedience to orders. Sensation, motion, and taste were perfect; hearing and smell were rather weak. She improved while in the asylum so far as to be able to do some work, and to abandon some foolish and destructive habits, to which she had become accustomed. In the second year of her residence she died of phthisis. The necropsy gave the following result:

The cerebrum weighed $35\frac{3}{4}$ ounces avoirdupois; the convolutions were small, with few tortuosities, and the sulci were shallow; the frontal lobes were imperfectly developed in proportion to the other parts of the brain. The corpus callosum was completely wanting, as were also the septum lucidum and the anterior and soft commissures. The olfactory nerve was absent on the right side, and was merely rudimentary on the left. The auditory filaments were slender; the cortical and medullary substance of the cerebrum and cerebellum were anæmic.

Atrophy of Cord.—There is comparative absence of nerve-tubes, and presence of amyloid bodies. It is in the cord even more than in the brain that we see in a striking manner the difficulties of

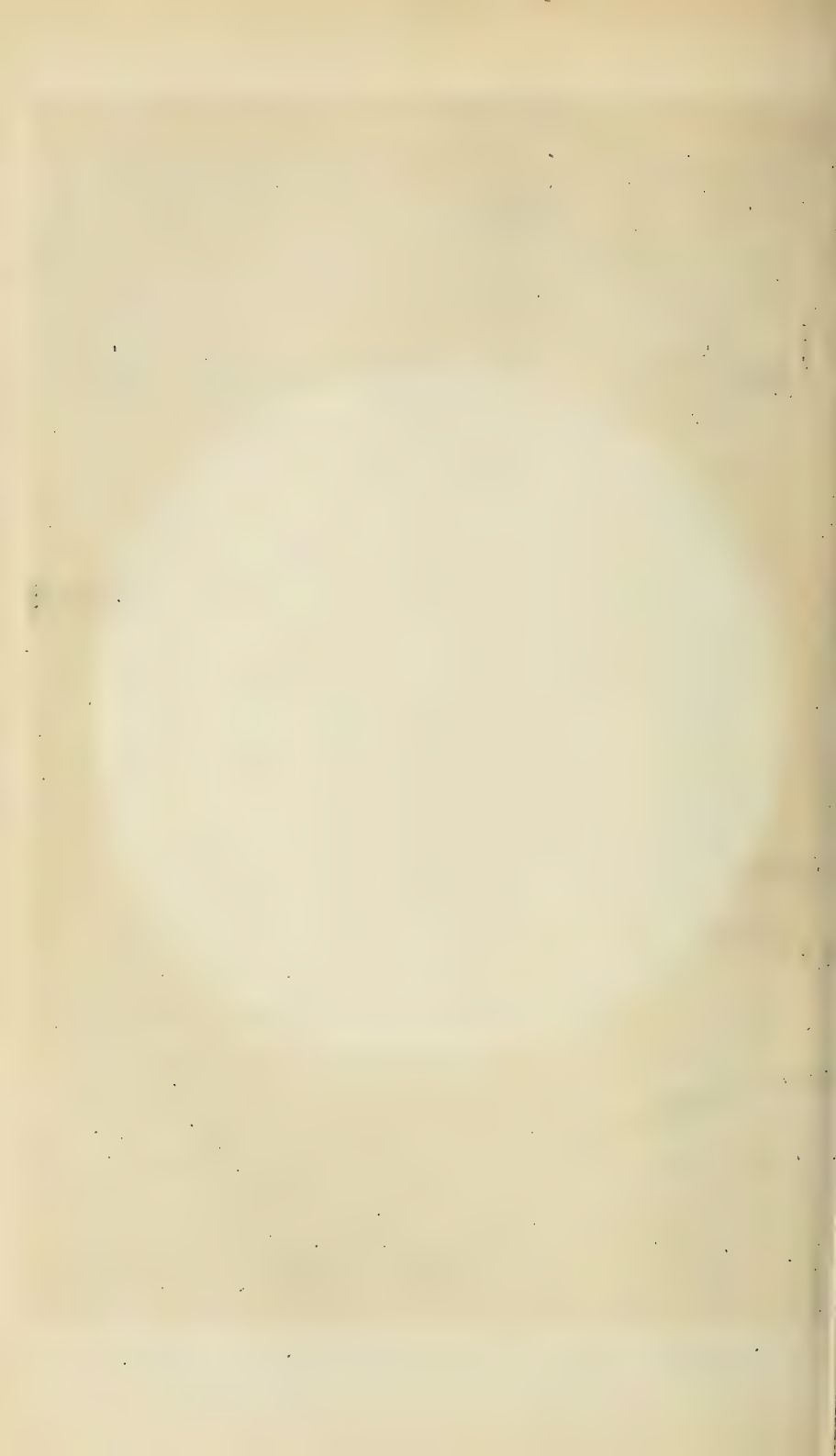
pathological distinctions. It is perfectly true that atrophy of the cord may exist *per se*, depending on atheroma of the capillaries, and manifested by a tolerably universal shrinking of the whole organ. This is seen most decidedly in the upper portion of the cord, as the condition is due sometimes to lesions of the brain that have practically cut off much of the use of the cord below them, or to lesion of the cord that has a similar effect on the part of the cord lying further down. The white matter of the cord is solely or mainly affected.

But the more common varieties of atrophy of the cord are due to other conditions, to which the atrophy is merely the sequence. We may meet with atrophied appearance of the cord in cases of general or localised sclerosis, when disseminated sclerosis attacks the whole thickness of a cord in various portions of its length; when it confines itself to one column or set of columns; when sclerosis in the non-disseminated form attacks a small portion of the cord; when grey degeneration affects a spinal region, the posterior columns for instance, or the groups of cells in the posterior cornua; and lastly, we see it in great perfection in some cases of chronic myelitis, as in Plate 4. In one case observed the calibre of the cord on transverse section did not exceed half what is normal, and this, too, in its whole length. Much more frequently, even where atrophy depends on chronic myelitis, it is partial. In the case above alluded to almost all the nerve-structure had disappeared.

In the other forms the loss of nerve-tube has depended on the pressure consequent upon the sclerosis, the contraction of the hyperplastic connective tissue.



x 20



Atrophy of the cord, therefore, should lead to further investigation. It will then be found to depend either on atheroma of the smallest vessels, or myelitis most usually chronic, or grey degeneration, or general or partial sclerosis, disseminated or non-disseminated.

The so-called hypertrophy of the brain is merely an extreme hyperplasia of the connective issue. There is no real hypertrophy of nerve-cells or nerve-tubes. It is pretty much the same condition as sclerosis, only in the latter there is retraction of the connective tissue and consequent destruction of the nerve elements. In hypertrophy of the brain the retraction has not taken place, and the nervous elements exist in their normal amount. Localised hypertrophy of one hemisphere of the brain may coincide with atrophy of the opposite side of the body.

Rokitansky states, as the result of many microscopical examinations, that its augmented bulk is not produced by the development of new nervous fibrils, or by the enlargement of those already existing, but by an increase in the intermediate granular matter, most probably due to an albuminoid infiltration of that structure. This is doubtless but the first stage of the process of connective tissue growth. Neither the base of the brain nor the cerebellum is liable to this affection.

Softening.—For the production of softening the most important element is suppression of the circulation; and the varieties of softening depend more or less on the nature and mode of this suppression. Obliteration of the arteries, of the capillaries, of the sinuses, will form the main causes of cerebral softening.

It may be due to inflammation, to hæmorrhage, to infiltration of serum, to the neighbourhood of tumours, to interruption to the course of the blood by embolism, by thrombosis, by disease of vessel such as atheroma, not necessarily leading to thrombosis, by artificial means, such as ligature of vessels, &c. But all these conditions own a common point—the suppression, total or partial, of the circulation. The stasis or the thrombosis existing in inflammation, the rupture of vessel in hæmorrhage, and the consequent cutting off of the circulation beyond the seat of rupture, the pressure of a tumour on the surrounding vessels, may all either lead to, or imitate in their effects, the embolic or thrombotic obliteration of vessels to which softening is due.

In inflammatory softening new formations exist. Connective tissue is generated; an exudation of albuminous fluid containing fine granules takes place, and, mixed with red globules, forms a reddish pultaceous mass. The pulpy mass undergoes partial absorption, and is replaced by a white turbid fluid. There are often also small hæmorrhages met with.

Jaccoud's remarks on softening are so pertinent that I am tempted to give you a free translation of them.

Partial anæmia and softening by necrosis of the cerebral regions nourished by the obstructed artery are, in chronological order, the two phenomena which follow the interruption to the course of the blood. When death occurs before softening takes place, there is no need to look for an abnormal white colouring to recognise local anæmia; several circumstances may mask or efface all difference of colour between the anæmic part

and the neighbouring regions ; when an arterial branch is obstructed, a collateral or compensating circulation is developed in the vessels that have remained patent ; this afflux of blood produces hyperæmia of the tissue which surrounds the anæmic sphere, and this congestion, extending itself nearer and nearer by the capillaries, causes the ischæmic colour to disappear. On the other hand, the obliteration of the artery is frequently followed by that of the corresponding venous branches ; thence a congestion by stasis, the deep red hue of which masks the anæmic pallor. Consequently it is well not to trouble oneself with the illusory appreciation of colour, and only to judge of the partial anæmia by the state of the arteries ; we shall find the occlusion ; and the examination of the walls, the coagulum, and the heart will allow us to decide whether one has to do with a spontaneous obstruction or an embolism.

Softening is circumscribed in one or more strictly limited spots ; it results from the suspension of the nutritive process in the parts which were nourished by the obliterated vessel ; in its mechanism and its cause this softening does not differ from the mortification produced in all organs by the uncompensated obstruction of a nutritive artery ; thence the very good name of 'Cerebral Necrosis,' which is generally given to it to distinguish it from all other kinds of cerebral softening. With the exception of the very rare cases in which death is so rapid that the centre of the necrosed spot is found white and bloodless, softening presents three stages characterised by a special colour ; the first is the stage of red softening, the second of yellow, the third of white.

Red softening is only developed 36 or 48 hours after the occlusion of a vessel : during this interval the collateral circulation is established ; and if it is sufficient to prevent the death of the tissue, all the ulterior accidents may be avoided. This stage is characterised by a diminution in the consistence of the tissue, and a colour which varies from a rose to a deep red ; the tint is more intense at the circumference of the spot, where the vessels are strongly dilated and hyperæmic.

It is not only a simple congestion which is produced ; often the small vessels are ruptured under the influence of the increase of pressure, and we observe here and there in isolated or confluent spots small punctiform hæmorrhages which give to the tissue the aspect of a reddish pulp ; in other cases, the external appearance being almost the same, there has been no true hæmorrhage ; the vessels are not ruptured, but the increase of pressure has caused the transudation of a serum, coloured red by the dissolved hæmatine : hence a pseudo-hæmorrhagic imbibition, which contributes in a great degree to the separation of the histological elements and to softening of the spot. Under the microscope the cells and the nerve-fibres are separated : the most part present still their normal characters ; some may be already ruptured and granular. Except in the case of hæmorrhage, the capillaries are intact or filled with coagulated blood. The duration of this stage varies from 8 to 14 days.

In the stage of yellow softening, the contents of the spot have already the aspect of a 'bouilli' more or less thickened ; the fibres and the nerve-cells are

ruptured, separated, granular, and in course of retrogressive or fatty metamorphosis; they are often even scarcely recognisable; the walls of the vessels are covered with fatty granulations; they contain the elements of the blood, and particularly elements of free hæmatine and white globules; these degenerated leucocytes and fatty granulations constitute the corpuscles described by Gluge under the improper name of inflammatory corpuscles, or granular bodies of inflammation. It is these elements that give to the spot its characteristic yellow colour.

White softening is only effected after several months; the contents of the spot, perfectly white, appear as a milky pulp more liquid than solid, in which are suspended whitish flocculi. The nerve elements, the blood-globules, the capillaries have disappeared: the microscope shows nothing but numerous granulations, drops of fat, and granular cells, so that the liquid presents an accurate resemblance to colostrum.

When the spots are not too considerable, absorption is possible; it often leaves after it a depressed cicatrix or a small lacuna, a persistent vestige of a former pathological condition. This is the most complete mode of repair; in other circumstances absorption is not complete, but a cyst is formed, which isolates the spot from the healthy tissue. These various curative processes represent the fourth stage of necrosed softening.

In certain cases the examination of the brain demonstrates plainly a spot of necrosis, but the obstructing clot cannot be found. As this difficulty

only presents itself in old-standing cases, we must admit, with Bamberger and Hasse, a complete absorption of the clot or a total atrophy of the artery. (Meissner.)

The spots of cerebral necrosis often coincide with similar lesions of other viscera, especially the spleen and the kidneys.

For the occlusion of an artery to result in necrosis, it is necessary that a sufficient collateral circulation should not be immediately established ; it is necessary, therefore, that in the case of the anterior cerebral regions the obstruction should be situated beyond the circle of Willis. Observation verifies this presumption, and the obliterations of the internal carotid, for instance, have not the effect, unless the clot should have prolonged itself by successive deposits beyond the arterial hexagone : when this prolongation has not taken place, the cure may be complete and rapid. This condition applies equally to thrombosis and to embolism ; but whilst the former has no situation it specially prefers, the second is more frequent in the left Sylvian artery than anywhere else. Cohn pretends even that the obstruction only occupies the right Sylvian artery in the cases where the embolus does not come from the heart, but from the brachio-cephalic trunk, or from the right carotid. This assertion is too absolute ; for in the table of Meissner I find eight cases of right embolism in which the heart was the sole cause. In obstruction of the Sylvian artery, the infarctus generally occupies the median spots of the corpus striatum ; the region of the hemispheres under the





X 300

Micrograph, 1912.

Hanks & Co., Inc.

SOFTENING OF PONS

immediate control of the collateral circulation remains intact. When the choroidal artery is obstructed, the softening is situated in the white substance of the hemispheres. The obliteration of one vertebral only has no effect, because of the afflux through the trunk of the basilar; but the infarctus remains persistent when the basilar or the posterior cerebral is the seat of the obstruction. In one case of Brunnicke, the occlusion occurring in the trunk of the basilar, the softening occupied the pons and the optic thalamus. In a case of Bennett, with thrombosis of the same artery, the necrosis was confined to the pons. In a case of Cohn, where the posterior cerebral was occluded, the infarctus occupied the optic thalamus, and did not reach the corpus striatum.

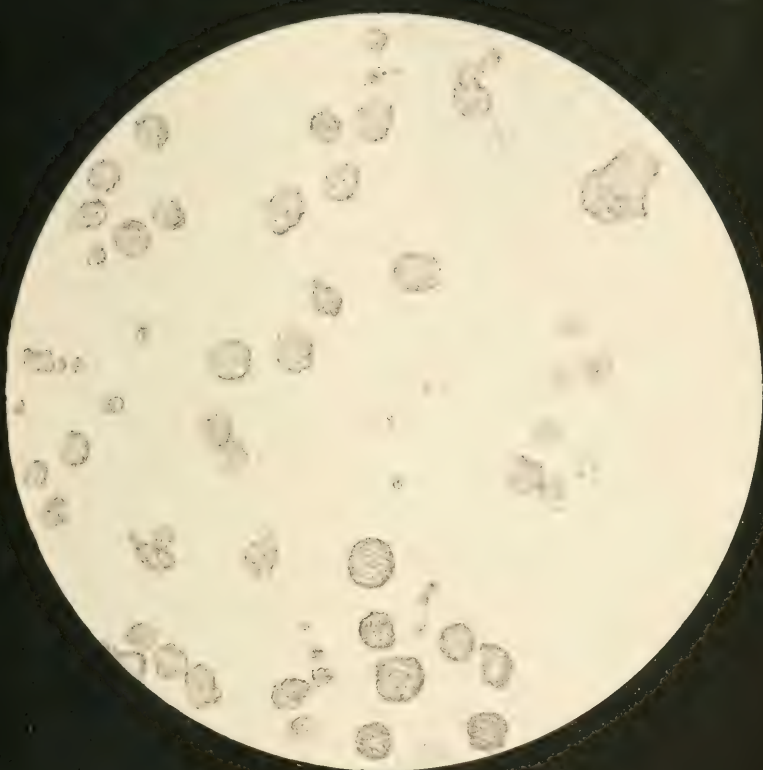
The case from which plates 5 and 6 were taken was this:—

William G., aged 30. Has a tuberculated syphilitic rash all over the body, and ulcers on the legs. Partial right hemiplegia, but no impairment of speech. Soon after admission his intellect began to be impaired and his speech thick. Later he became temporarily hemiplegic on the left side, this symptom lasting only a few days. About two months ago he suddenly became blind, and this amaurosis lasted for several days, and then passed off. The right hemiplegia continued. Some paralysis of bladder. A state of stupor very gradually came on, which terminated in death.

P. M. Ex. Broca's spot and every part of both cerebral hemispheres quite healthy. The pons was

the seat of softening over a space the size of a filbert, occupying rather more of the left half than of the right, but passing a good deal over the middle line on the right side. The nervous tissue was here quite deliquescent. The vessels of the circle of Willis a little fatty. All the other organs healthy, except that in part of the left lobe of the liver there were regions of yellowish discolouration. On microscopical examination, after preparation, the blood-vessels were the only structure left, except a few nerve-cells and fibres atrophied and isolated by the disease. But there were also found some strange round bodies, about $\frac{1}{1000}$ of an inch in diameter, that seem to be conglomerate cells. Plate 6.

Obliteration of the Capillaries.—This subject is very imperfectly known, for the question of the pathological anatomy is alone elucidated. The obstruction of the capillaries is spontaneous or embolic. The former—capillary thrombosis—is connected with lesions slow and gradual in their progress, which have been carefully studied since the first works of Paget and Robin. The chief of these lesions are the deposit of fatty granulations in the walls; in aged individuals, moniliform dilatations and atheromatous and calcareous incrustations of the small cerebral arteries. As to dissecting aneurism, we look upon it as a secondary lesion, consecutive to necrobiotic softening, and not as the primary pathogenic condition of the alteration of the nervous tissue. Whatever be the preliminary lesions of the capillaries, they have for their effect retardation, and then arrest of the course



X 950

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CELLS FROM SOFTENED PONS.

of the blood; and, finally, the ischæmic softening of the histological territory fed by the diseased vessels. The initial characteristics of these softenings are not known. The spots are small and multiple in direct ratio with the diffusion of the lesions. The course of the process, always slow, differs absolutely from the embolic necrobiosis described above, and is rather related to softening by disseminated thromboses. It is seldom that the spots are situated in those cerebral regions which, impatient of all lesion, show the least alteration by symptoms clearly appreciable. It is in the white mass—in the cortical layer of the hemispheres—it is in the centre of the corpus striatum, that we most generally meet with them; and unless the spots of softening are numerous, the lesion remains without symptom, and is recognised only *post mortem*. The obstruction by embolism forms two classes, according to the nature of the embolus. The one class act only mechanically, merely as obstructing bodies, and producing only the ordinary lesions of ischæmic necrobiosis; the other (specific embolisms) owe to their original seat specific properties that they transport with them, so that wherever they are arrested they provoke lesions of the same nature as those of the primary spot. It is not yet well established that simple or mechanical embolism may be effected by pieces of granular or fibrinous material originating from clots in process of breaking down; consequently, this first class of embolisms should be limited to three varieties, which are pigmental, calcareous, and fatty embolisms.

The pigmental embolism can only be produced when the blood contains an abnormal quantity of pigmental corpuscles; that is to say, in the state known under the name of melanæmia. This state has so far only been seen in persons affected at the time, or who have been previously attacked with intermittent fever. These fevers are the sole cause both of the melanæmia and of pigmental embolism. It is of no great importance at the moment whether the pigmental corpuscles with which the blood is then charged originate exclusively in the spleen, or also in the liver. What is certain is, that these cellular or cylindrical granulations may accumulate in the cerebral capillaries, and produce obstruction in them, either directly or by the coagulation of blood they induce. This obliteration prefers as its seat the cortical layer of the hemispheres. When it is general, the change of colour which it produces may be appreciated by the naked eye. The substance is of a dull grey, and the consistence is diminished. It is only exceptionally that we find grey tracts in the white substance. Small capillary hæmorrhages coexist sometimes with this obstruction, and in two cases Frierichs has observed a meningeal hæmorrhage. Latent as regards symptoms when it is very limited, pigmental embolism reveals itself in other cases at the beginning by headache, hallucinations, delirium, convulsions; that is to say, by the phenomena of excitation, and, eventually, by a coma more or less profound. Paralysis is extremely rare; it may be partial, or hemiplegic, or paraplegic in form. These symptoms have nothing characteristic

of themselves ; they only acquire significance by the knowledge of the antecedents of the patient, by the swelling of the spleen or of the liver, and by the greyish brown colour of the external integument. It would be an error to refer constantly to a pigmental obstruction, the cerebral symptoms of the intermittent fever called '*accompagnée céphalique*.' Even when this form of fever is very clearly characterised, the capillary embolism may be wanting. It was absent, indeed, six times in twenty-eight cases observed by Frierichs. On the other hand, though the cerebral accidents are vigorously intermittent, it is very difficult to attribute them to a fixed lesion of the same kind as that which occupies us. Sulphate of quinine in large doses, cold applications to the head, cutaneous revulsions, are the basis of treatment ; but the prognosis is always serious.

Calcareous embolism is characterised by a calca-reous incrustation obliterating the capillaries. The cerebral tissue resists when cut, and the vessels appear on the surface of the sections rigid, like the hairs of a brush. This alteration, always limited to the smallest vessels, often coincides with similar lesions in the lungs and the mucous membrane of the stomach. According to the theory of Virchow, I place this obliteration among the embolisms, and not among the spontaneous obliterations. According to him, we have to do here with calcarcous metastases. The reabsorption which takes place in the bones throws into the blood these mineral elements, which, in consequence of unknown causes, are deposited in the parenchyma of

organs, instead of being eliminated by the organs of secretion. Over and above this, nothing is known touching the effects of this obstruction. It is probable by analogy that it also provokes small centres of softening, but it is all obscure. The same thing may be said for fatty embolism, which is demonstrated as a fact by some observations, but the anatomical and clinical facts of which are wholly unknown.

In the specific alterations the embolus is formed by pus or by the débris of organic matters in decomposition carried from gangrenous, putrid, or purulent centres. Virchow admits that these débris, once arrested, produce by catalysis some modifications similar to those which go on in the original centre. Panum believes rather that the decomposition of these débris continues after their arrest, and that the products of this decomposition are a cause of irritation to the contiguous tissues ; whence inflammation, suppuration, and sometimes gangrene. Whether one adopts the idea of the similar production, or that of irritation of the neighbouring parts, the chief fact is this : the embolisms are the causes of the spots and of the abscesses called metastatic. For the particular case of the brain, see what takes place. Some fragments detached from a putrid or a purulent centre arrive in the capillaries (or the smallest arteries), obstruct them, and provoke lesions and symptoms which have been described as the cerebral accidents of the putrid or purulent infection. I do not pretend that it is not so, but I believe that we must observe that the embolus, when it does not come from the lung, passes through a

course truly enormous. If the primary centre is in the spleen (of which there have been examples), we must admit that the embolus traverses the liver, the right side of the heart, the pulmonary artery, the lung, the pulmonary veins, the left side of the heart, the carotid, to escape at last, after so many peregrinations, in the cerebral capillaries. The thing is possible, but it 'demands a little complaisance,' the more so that here the mechanism invoked cannot be demonstrated by fitting together the two fragments of the clot, as in the ordinary embolism, so that we must admit many lesions at one and the same time, without reciprocal mechanical relations; lesions governed and provoked all of them by the general morbid disposition. As the observations are not sufficiently clear to dissipate all these doubts, an absolute solution is not possible. Already O. Weber, whilst admitting these diffused capillary embolisms, rejects the conclusions of Virchow, Panum, and Cohn on their specific nature, and only attributes to them a mechanical rôle.

Obliteration of Venous Sinuses.—The primary obliteration is generally situated in the superior longitudinal sinus, and thence extends symmetrically into the neighbouring sinuses (the lateral, &c.); if it is secondary, it naturally occupies the sinus nearest to the pathogenic lesion. The sinus is turgid, tense, and filled with a fibrinous clot; this clot is resisting, homogeneous, non-adherent, formed of concentric layers, if it is not old; later it is softened in the centre, where one finds a puriform 'bouilli,' which for a long time was taken for pus, and which is composed of fat, breaking down

granulations and leucocytes. Unless the clot should be of inflammatory origin, the adhesion to the wall is slow ; if it results from the formation of connective tracts, the membranes of the vessel are intact. In secondary thrombosis, which most frequently succeeds to caries of the cranial bones (30 times in 39 cases of Lanceraux), the walls of the sinus are altered at the level of the diseased bones ; they are thickened, friable, with small resistance ; sometimes even they are destroyed ; the coagulum is not always composed of pure fibrin ; it contains besides some pus and débris of false membranes.

The cerebral lesions produced by obliteration of the sinuses result from the increase of pressure in the capillaries and the afferent veins, or rather also from the arterial ischæmia, caused by the venous stasis ; these are capillary hæmorrhages, interstitial or meningeal, œdema of the cerebral tissues, and of the pia mater, with or without dropsy of the ventricles and superficial centres of softening, which occupy symmetrically the surface of the middle lobes, and are found especially in the grey substance. Some lesions, purely inflammatory, of the meninges (false membranes, pus) often coincide with thrombosis from caries of bone.

Depôts of softening, which lie within the hemispheres, cannot be obliterated, because their walls cannot collapse, and thus arises a cyst. Near the surface, however, either of the brain or a ventricle, there may be a slight depression of the separating layer, and the layer of softening is completely obliterated (its place being taken by cerebro-spinal fluid). Rokitsky

mentions a special form of yellow softening, consisting of sharply circumscribed spots, in which the cerebral substance is connected with a very moist, tremulous, and occasionally gelatinous pulp, of a straw-yellow colour; the expressed fluid is acid. He thinks it is a peculiar chemico-pathological transformation of brain substance in which the liberation of an acid, phosphoric, or one of the fatty acids may be considered to be the important phenomenon.

Calneil records a very interesting series of cases of cerebral softening from various causes, under the title of chronic cerebral softening, or chronic local encephalitis, without clots of blood.

Of 27 cases, in 6 the cranial bones appeared injected, in 1 perforated, in 1 with exostosis.

In 4 the cerebral dura mater was injected, in 5 strongly soldered to the bony tissue, in 2 altered by accidental products.

In 6 the cavities of the cerebral arachnoid contained serum, in 3 the cavity of the right arachnoid contained false membranes, in 2 some kind of plastic plug, in 1 a cyst filled with blood.

In 7 the pia mater of the right cerebral lobe was injected, in 13 infiltrated with serum, in 4 thickened, in 8 adherent to the convolutions locally inflamed.

In 9 the pia mater of the left cerebral lobe was injected, in 15 infiltrated with serum, in 3 thickened, in 5 partially adherent to the subjacent substance.

In 6 the convolutions of the right cerebral lobe showed spots disintegrated and pulpy, in 5 spots disintegrated with mixture of cellulosity, in 3 spots indurated

and shrivelled, in 3 faded and atrophied cores, in 4 false membranous spots of orange colour, in 2 spots of ecchymosis.

In 4 the convolutions of the left cerebral lobe showed spots in a state of ecchymosis of a strawberry colour, in 4 spots disintegrated and pulpy, in 3 spots disintegrated with a mixture of cellulosity, in 3 false membranous spots of an orange colour with softening, in 3 without softening, in 5 spots indurated, faded, and shrivelled.

In 3 there were in the depth of the right cerebral lobe spots of softening in a creamy state, in 1 spots softened and mixed with cellulosity, in 3 spots with cellular bands, in 1 cavities with softened walls, in 1 with consolidated walls, in 2 spots of a raspberry colour. The white substance was often injected, sometimes yellow, sometimes firm and resisting.

In the depths of the left cerebral lobe there were found in 4 local softened spots, in 5 spots filled with cellular band-like products, in 1 hollow spots with cellular walls. In 10 the white substance of this lobe was found injected, in 3 generally indurated.

In 3 the right corpus striatum was found faded and shrivelled, in 5 locally softened, in 3 hollowed with cellular cavities.

In 1 the left corpus striatum appeared stunted, in 2 softened locally, in 3 red-coloured in places, in 4 traversed by cellular bands.

In 1 the right and left optic thalamus were stunted and indurated, in 1 disintegrated at their surface.

In 4 the lateral ventricles were distended by serum ;

their walls were traversed by vascular arborizations of great intensity in 3; in 4 indurated, in 2 disintegrated and softened.

In 3 the central parts, especially the septum of the ventricles, were softened, in 5 indurated, and as it were atrophied.

In 4 the pia mater of the cerebellum adhered to the surface of the hemispheres of the cerebellum, in 6 it was injected.

In 6 the superficial grey matter of the whole cerebellum was softened, in 4 very red-coloured, in 2 yellow.

In 1 the two lobes of the cerebellum were atrophied, in 1 there existed on the right side a spot of ecchymosis, in 1 a false membrane, in 1 a spot of softening, in 1 on the left side a hollow spot with soft walls.

In 2 the pons showed in its centre a spot or several spots in a cellular state, in its right half a cellular spot once.

In 3 it was injected, in 1 soft, in 2 hard, in 1 shrivelled.

In 1 the medulla oblongata was diminished in firmness, in 1 it was too firm, in 1 pierced with small cellular spots.

In 4 the spinal cord was thin and indurated, in 1 softened and disintegrated, in 1 of a violet colour.

In 14 a microscopical examination was made, both of the products contained in the cavities of the cerebral arachnoid, and of the products contained in the different spots of local encephalitis.

Softening of the spinal cord is due to injury, or inflammation, or to the pressure of a tumour. Softening

is sometimes of the whole extent, generally of some spot, or of several spots with healthy spaces between, especially of the posterior columns. The affected parts may be swollen or atrophied. There is no special alteration of the arteries.

The grey and white matter seem to be blended homogeneously, as Hammond says, and the double crescentic arrangement of the grey matter is lost.

Microscopically, the nerve-tubules are broken up; oil globules and granule masses (the constituent of which is fat) have taken their place. In the grey substance the nerve-cells are destroyed, and oil and fat appear in large amount. Even the neuroglia exhibits a similar disintegration and regressive metamorphosis.

The areas of softening, due to a kind of disseminated myelitis, are sometimes well seen in fatal cases of paraplegia after small-pox.

Grey Degeneration.—The terms grey degeneration and sclerosis are used variously by different writers. To some they express different ideas; to others they are one and the same thing. It is certain that the connection between them is very close. The colour is more or less common to both, and is due to the loss of the sheath of the white substance of Schwann on the part of the nerve-fibres. Rindfleisch indeed classifies both under one heading, speaking of grey degeneration as taking two forms—1, simple non-inflammatory, grey degeneration proper; and 2, indurating inflammatory form, sclerosis.

In a similar manner also Gintrac speaks of patches of grey or amber-yellow colour, a sort of half transparency of the affected parts, and says that the diseased portions may be indurated or softened.

It may affect various points or regions in the brain and spinal cord, or the cord only, the lateral and anterior columns, or the posterior columns of the cord, the most frequent seat of this lesion. The breadth of the disease is generally greater than its depth.

It may extend the whole length of the cord, or it may be interrupted by portions of healthy spinal cord tissue. The posterior roots of the spinal nerves are very often implicated; they are atrophied, and are themselves in a state of grey degeneration.

Microscopically we find amorphous matter, finely granular, between the nerve-tubes, disappearance or atrophy of the latter, numerous nuclei of connective tissue, amyloid bodies, and fatty granulations.

Rindfleisch says the first factor of this grey degeneration is an increase of the interfibrillar connective substance of the white fibres; the second factor, the disappearance of the nerve-fibres in the attacked columns (probably by atrophy caused by pressure of the increased connective tissue); the third factor, increased connective tissue surrounding the small vessels, with fatty and pigmental granules on them, and a lustrous condition of the vascular walls; the fourth factor, corpora amylacea, the nature of which is unknown. They are probably due to an amyloid infiltration of sound neuroglia cells.

The disease is either due to a chronic irritation or to a perversion of nutrition, causing a loss of the normal condition of the nerve-tubes. The tubes perish, and their place is taken by hyperplasia of connective tissue and these corpora amylacea. These

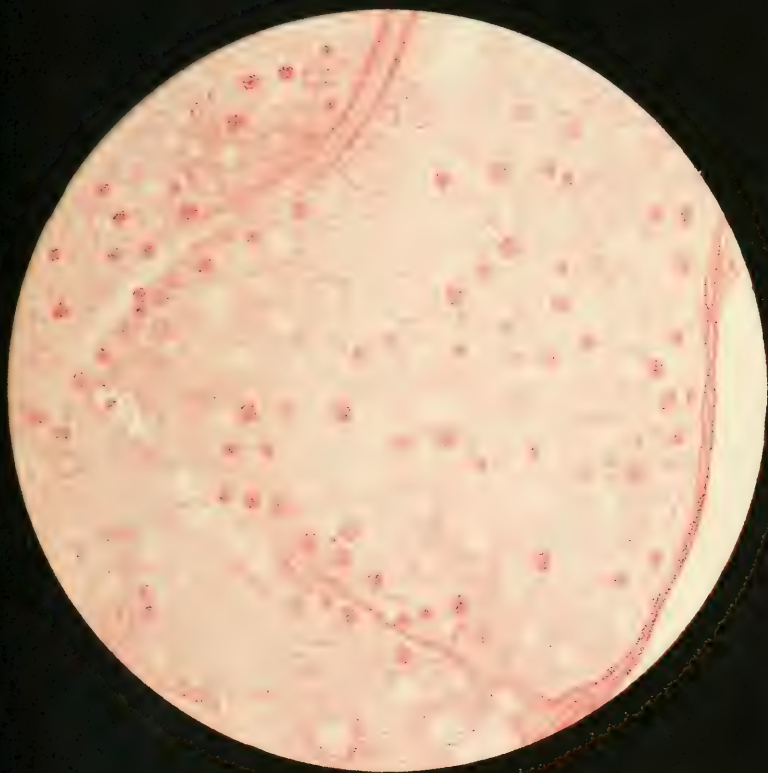
amyloid bodies exist in far larger amount than in true sclerosis, whilst the hyperplastic condition of the connective tissue is much less marked than in the latter lesions. Still the difference is very little more than one of degree, and the two lesions can scarcely be separated pathologically. Whilst, however, the distinction between them pathologically is difficult to draw, there is certainly a difference, on microscopic examination, between a well-marked instance of grey degeneration and one of complete sclerosis.

Plate 7 is a tolerably good example of grey degeneration affecting the cord. In this case the whole of the cord was more or less affected, and in its whole length. But the posterior columns and a small portion of the grey matter were chiefly disintegrated, and that mainly at the cervical and lumbar swellings.

Plate 8 represents the large amount of amyloid bodies found in this lesion, and shows also some increase of the connective tissue.

Grey degeneration, of the cervical swelling specially, of the cord is sometimes accompanied by progressive atrophy of the muscles, a loss of their transverse striæ, with granulations and fatty globules, and a recent connective tissue.

When the lesion is located in the posterior columns of the cord, it is associated with loss of co-ordinating power. This condition is very often accompanied also with a similar degeneration of the optic tract or centre. A condition standing mid-way between grey degeneration proper and sclerosis is sometimes found in the cord, depending on atheroma of the capillaries. There is



X 450



X 300

degeneration and atrophy of a greater or less number of nervous filaments, the formation of granular corpuscles in the degenerated tissue, and proliferation of the connective tissue which takes the place of nerve-tubes. In these cases grey degeneration, sclerosis, and softening of cord seem almost to merge one into the other.

Sclerosis.—Speaking in general terms, sclerosis, wherever met with, is composed of two conditions, the latter of which depends on the former; the first being the abnormal development of the connective tissue, the second the compression and gradual destruction of the tissue of the part by the pressure and contraction of this proliferated connective tissue. Although the pathological anatomy of this morbid condition is the same, however it be distributed in the nervous centres, it is convenient to speak of it as taking three forms: 1, general sclerosis, seldom affecting the brain, but not unusually affecting large tracts of the cord; 2, disseminated sclerosis, ‘*sclérose en plaques*,’ which is found both in the brain and spinal cord; and lastly, a form which may be only a variety of disseminated sclerosis, the miliary sclerosis, described by Kesteven, Tuke, Rutherford, and other British observers. Benedikt, however, says that it is an error to speak of ‘*sclérose en plaques*,’ since it has been established by Bourneville’s researches that the disease is not limited to foci, but that even the apparently healthy interstitial parts are diseased. We have, he says, in reality, to deal with a diffuse neuritis, which derives its character, at least so far as regards its spinal symptoms, from a combination

of disease of the anterior or lateral columns. Before we come to the distribution of this abnormality, it is only fair to tell you that pathologists differ as to the interpretation of the first element of sclerosis, because physiologists differ as to the existence of connective tissue at all in the nervous centres. Some physiologists, such as Virchow, assert the existence of a tissue of this kind, which they call the neuroglia. In health it is so fine that its demonstration is almost impossible. Analogy is certainly in favour of its existence. It can be demonstrated in the liver, spleen, kidney, mucous membrane of the stomach, &c., to say nothing of many tumours. To those who believe in its existence, sclerosis is in its first element a simple hyperplasia. To those who will not acknowledge that neuroglia is a healthy constituent of nervous-centre tissue, sclerosis is a new formation. Out of a good deal of controversy, in which various opinions have been held by Virchow, Bidder, Henlè, Stilling, Deiters, Kölliker, Boll, Jacobouritz, &c., to say nothing of British observers, it seems now to be pretty generally allowed that there is in the nervous centres an element that answers to a connective tissue; though whether this is formed by the union of the processes of the spider-web cells of Deiters and Rindfleisch, or in some other way, is still a matter of controversy. But whether this nerve-cement, as it has been called, is nominally finely granular or finely fibrillated—and each view has strong and learned adherents—it is certain that the first step in sclerosis is the transformation of this cement into a finely-meshed network. The only anatomical distinction that can be

drawn between sclerosis and grey degeneration is in the greater or less proliferation of this fibrous network, and the fact of the presence of corpora amylacea in grey degeneration. It is wholly a question of degree, and I am loth to assent to the one being inflammatory and the other not. It is probable that both are of inflammatory origin.

Rindfleisch, speaking of the grey foci from which sclerosis starts, and the smallest of which are about the size of a pin's head, says that an attentive examination of the smallest of these grey foci leads to the curious discovery that they have all got a red spot or line in their centre, a distended blood-vessel cut across transversely or obliquely. The microscope shows that all these vessels, together with their finer ramifications, are in a state which we should not scruple to call one of chronic inflammation. He says that in these alterations of individual vascular tufts is the first anatomical element of the disease, the second consisting in a fibroid metamorphosis and overgrowth of the neuroglia.

Van der Kolk thought that sclerosis was due to repeated congestions, which lead to the exudation of an albuminous fluid.

It may affect the brain in a variety of ways. Gintrac speaks of it as affecting several parts of the brain at once, or one cerebral hemisphere, or the anterior lobes of the brain, or the posterior or the middle, or the corpora striata, optic thalami, and cornua Ammonis, or the cerebellum, or the pons and the medulla oblongata, or various parts of the spinal cord, or very rarely the nerves. The optic nerve is

more liable to this lesion than others ; and in specimen 765 of the Oxford Pathological Museum may be seen the trunk of the facial nerve as hard as cartilage. There was ulceration round it in the internal ear, and at its exit from the bone it came to an end in consequence of the ulceration, so that all communication with the face was cut off.

Not unfrequently the sclerosed regions will lie subjacent to the ependyma of the ventricles, and then the ependyma will be thick and indurated ; and the walls of the ventricles beneath the ependyma may be rugose and full of small projections of indurated cerebral tissue ; and much fluid will be found in the ventricles. It may occur coincidently with softening, or be produced round an old spot of hæmorrhage, or coexist with a loss of substance of the brain, more or less considerable.

Pinel states that it does not invade the grey matter of the brain, but this is certainly a mistake. Not to mention all the authorities quoted by Jaccoud, this condition has been found in one case in the hemispheres, centrum ovale, arbor vitæ of cerebellum, pons, and anterior and lateral columns of the cord ; also in the superior and inferior marginal convolutions and the borders of the fissures of Sylvius on both sides, with disseminated amyloid degeneration in the anterior and lateral columns, and proliferation of the connective tissue in the spinal ganglia and the cervical cord of the sympathetic ; also in the cortical and medullary substance of the hemispheres, the corpus callosum, fornix, optic thalami, corpora striata, pons, crura cerebri, cerebellum, medulla oblongata, lateral and anterior

columns of the cord, and a little in the posterior columns; also considerable induration of one hemisphere. Again, in a case mentioned by Hirsch, there were twenty spots on the left side, forty on the right in the medullary substance of the hemispheres, with the pons, medulla oblongata, optic thalami, and much of the grey matter of the cord similarly affected; whilst in a case mentioned by Liouville both substances of the hemispheres, the corpora striata and optic thalami, cerebellum, crura cerebelli, corpora mamillaria, pons, all the right olivary body, the olfactory nerves, the optic, the motores oculi, the left fifth, the left facial, and the spinal nerves were all in a state of sclerosis.

Is it ever general over the whole brain?

Dr. Quain has informed me of a case he had seen in which the patient, a young lady, fell into a state of quiet hebetude, and in which at the autopsy there was found great increase of the cerebro-spinal fluid and atrophy of the whole brain, with a very well-marked induration of all the brain-substance, both grey and white matter. No microscopical examination was made. But a case recorded in Bright's Medical Reports makes this point tolerably certain: 'A little girl, for the last twelve months of her life, became stiff and incapable of the slightest effort. The thumbs were drawn in and the feet stretched out. There was difficulty of swallowing, and apparently the power of vision was gone, and she became quite imbecile. The skull was well formed. On removing the dura mater about five ounces of fluid escaped, but it was impossible to say whether it was external or beneath the arachnoid

(probably the latter). No deposit on the dura mater, and the arachnoid not thickened. The whole volume of the brain was contracted ; on making a horizontal section, the knife met with remarkable resistance as soon as it had passed through the cortical substance, as if cutting soft cartilage. The section was very remarkable-looking. The corticle part was thick and pulpy ; the medullary part was hard, constricted, and shrunk, so that it seemed to enter the cortical substance like white bands. It could not be scraped as an ordinary brain. The margin around it was more prominent and whiter than the rest. When pressed on externally, the grey matter gave way, and the convolutions of white matter were felt beneath, like an irregular hard body, and by a stream of water the grey matter could be washed off, leaving the medullary convolutions exposed, like the rugose stomach of animals ; it appeared indeed more like a wax model of a brain. The ventricles were moderately distended and open, as if moulded in wax. The cerebellum had its cortical substance soft, but the medullary was hard, like the other ; but the corpus rhomboideum was soft, so that a cavity could be washed out of it. The medulla oblongata and spinal cord, as far as taken out, also hard.' Dr. Bright adds, this hardening was probably the result of chronic inflammation.

Perhaps the best description of sclerosis has been given by Dr. Batty Tuke, when speaking of this morbid condition as it is found in the brain and spinal cord of the insane. The microscopic appearances are as follows :

General Sclerosis.—Increase of granular material

of the outer layer of the grey matter of the brain. Normal condition of the two inner layers. Irregularity of the cells of the intervening layers as regards their number, shape, size, and distribution. Thickening and displacement of the nerve-fibres of the white matter. Increase of the neuroglia. Proliferation of the nuclei of the neuroglia and blood-vessels. Dr. Tuke gives a case in which there was hypertrophy of the right hemisphere depending on this condition, with atrophy of the left limbs.

Disseminated Sclerosis.—This Dr. Tuke would consider a synonym for grey degeneration. I have already told you that I believe grey degeneration and disseminated sclerosis vary only in degree; that in one the connective tissue proliferation is more than in the other; and that each owns an inflammatory origin of a low type. Dr. Tuke finds it among the cases of chronic insanity; most frequently in the white matter of the corpora striata and optic thalami in scattered patches of various size; less commonly in the white matter of the hemispheres, but, when there, in larger tracts. In general paralysis and in epilepsy this variety is often found in the medulla oblongata and the spinal cord, and frequently in an extreme form. The nerve-fibres are atrophied partially or completely; in transverse sections the axis cylinders and sheaths are destroyed, and the field is occupied by finely molecular and fibrillated material imbedded in a cloudy homogeneous plasm. In this matrix the proliferated nuclei exist, somewhat enlarged, sometimes slightly granular in appearance; but around the implicated spot they are to be seen in

much greater quantity, and not actually diseased. No fatty degeneration. The morbid plasm is probably modified neuroglia.

See Plate 9, in which the posterior columns show the chief increase of the connective tissue.

Miliary Sclerosis.—There is not necessarily any proliferation of nuclei. This condition is no way connected with blood-vessels. It is a disease of the nuclei of the neuroglia. It is common in both brain and spinal cord in the insane, especially in general paralysis and epilepsy. It may be described in three stages: 1st. A nucleus becomes enlarged and throws out a homogeneous plasm of a milky colour, displacing the nerve-fibres. In the centre of these semi-opaque spots a cell-like body is seen with a nucleus, being the original dilated nucleus of the neuroglia. The patch may be unilocular or multilocular. 2nd. The morbid plasm becomes distinctly molecular and permeated by fibrils. Further displacement of the contiguous tissues takes place, shown by induration of the compressed fibres and blood-vessels curving round the diseased part. After preparation in chromic acid the white matter shows a number of opaque spots, irregularly distributed, a molecular material with a stroma of exceedingly delicate colourless fibrils. They can be separated from the tissue and analysed; they are rendered transparent by strong nitric and sulphuric acid, the acid rendering the molecular matter fluid. (See Plate 15.) 3rd. The molecular matter becomes more opaque, and contracts on itself, the boundaries become



X275

puckered and irregular in outline, and the material often falls out of the section, leaving ragged holes.

What Dr. Tuke has thus graphically described as sclerosis found in the nervous centres of the insane can be seen to exist in a large number of cases in which the mental faculties have scarcely suffered at all—in spinal cord disease, not at all. It is seen in cases of myelitis in which life has been preserved for a few weeks. In a case in which the symptoms consisted mainly of intense neuralgic pain in the spine and limbs, whilst only very partial paralysis supervened towards the close of life, a longitudinal section of the cord showed in the grey matter some multipolar cells remaining, with the débris of tubes and broken-up tissue and many holes. The white matter was still more degenerated. Miliary sclerosis may be extremely multiple. Kesteven found spots of this degeneration in the brain of an idiot to amount to 25,000 to a square inch of surface. It is associated with amyloid and colloid bodies, and yet must be distinguished from them. The distinction is thus drawn by Dr. Tuke and Mr. Kesteven. Miliary sclerosis is distinguished from amyloid bodies by the polariscope, which, in the former case, will give neither the concentric rings nor the black cross of amyloid bodies, whilst it renders evident the molecular character of the degeneration. Colloid bodies (see Plate 18) may be distinguished by their clearly defined margins (miliary sclerosis presenting more or less irregular borders from the broken ends of fibrils, vessels, &c., encroaching on their space), and from their clear homogeneous translucent contents, which do not take the

carmine colouring. In extreme cases the appearance of sections containing colloid bodies may best be compared to a slice of sago pudding, for they exist in such large numbers as almost completely to fill the field of the microscope, separated slightly from each other by a fine granular material. They do not undergo the same gradation of development as miliary sclerosis, nor push aside the fibres, nor can they be removed as a separate substance from the dried section in which they exist.

Mr. Kesteven expresses surprise that the same lesion, miliary sclerosis, can be found in so great a variety of diseases. He has seen it in acute meningitis of cord, abscess of brain, locomotor ataxy, idiocy, leukæmia, chorea, tetanus, subacute myelitis, pseudo-muscular hypertrophy, paralysis with aphasia, hydrorachis interna, progressive muscular atrophy, apoplexy with hæmorrhagic softening, infantile convulsions, dementia, general paresis, malignant disease of the cord, glioma of the pons, internal hæmorrhage, and puerperal mania. In many of these conditions, however, there was inflammation, and in other instances the diseases were of importance, not so much *quâ* lesion, but *quâ* the seat of lesion; the physiological phenomena varying according to the part of the nervous centre attacked.

So little is definitely known about sclerosis, although its presence is now readily recognised after death, that I have thought it might help you to have a very brief analysis of Bourneville's and Guerard's observations on this morbid affection. They describe it as it affects the spinal cord alone, and the spinal cord with the brain;

and although their cases are not very numerous, their observations make a valuable addition to our knowledge of the subject:—

1. *The Spinal Form*.—Symptoms: debût sudden or gradual, generally gradual; ushered in by feelings of weight, formication, increasing weakness in one or both lower limbs.

1st period. The feebleness ‘paresis’ further attacks the upper extremities. This may show remissions and exacerbations. Progress painful. Help needed in walking. The patient staggers like a drunkard. After a variable time there are rhythmical tremors in the upper and lower limbs, only accompanying acts of voluntary movement. No tremor during repose. Sensation retained. No disturbance in the functions of nutrition, respiration, or circulation.

2nd period. To the paresis succeeds paralysis, which becomes more and more complete. Aggravation of all preceding symptoms. Soon rigidity and contraction of all the paralysed limbs, and attacks of tonic convulsion of the rigid limbs, especially the lower. The lower limbs are also the seat of spasmodic rigidity with intermissions of relaxation. The contraction affects first the lower limbs, then the upper, then the trunk.

3rd period. Motor power vanishes. Contraction is permanent. The patient becomes bed-ridden. Sensation generally good. Reflex power diminished or lost. Nutrition interfered with. Emaciation. Bed sores. Intelligence preserved all through.

The Cerebro-Spinal Form.—Debût sudden or gradual; either by sudden or progressive feebleness of one or

both lower limbs; or by this feebleness accompanied by cerebral or ocular phenomena; or by cerebral or ocular phenomena, followed by feebleness of the lower limbs.

1st period. Motor phenomena as in the spinal form. Feebleness of sight. Diplopia. Vertigo. Headache. Embarrassment of speech. Transitory apoplecticiform attacks without loss of consciousness. After a variable time, tremor of the lower limbs, then of the upper, then of the head, then of the eye, oscillation 'nystagmus,' then of the tongue. The nystagmus is generally binocular, but may be monocular.

2nd period. Aggravation of previous symptoms; then rigidity, or contraction, and convulsion, especially of the upper limbs, usually tonic, sometimes clonic, sometimes atrophy of papilla in retina. Speech worse. Sensation good. Intelligence gradually impaired; even hebetude sometimes. Respiration, circulation, and digestion normal, except that there is constipation. Urine healthy.

3rd period. Increase of spasm, rigidity, contraction, and paralysis. Speech unintelligible. Intense thirst. Swallowing difficult. Nutrition interfered with. Emaciation. Death from lung complications.

Our authors then endeavour to draw distinctions between this disease and some others, and in this attempt I think they fall into error. Their mistakes, however, teach us something.

Senile Trembling.—Disease of old age, whilst sclerosis is of middle age. Tremors almost permanent. Begins in head, then lips, chin, tongue, limbs.

Alcoholic Tremor.—At intervals ; worse in the morning than in the evening, diminishing after a dose of alcohol. Begins in hands, then arms, legs, tongue, lips. Speech hesitating, but not stuttering. Vertigo, insomnia, hallucinations, amblyopia, weakened intelligence.

Mercurial Tremor.—Only in workers in mercury. Vibratory movements. All the limbs attacked at once. No nystagmus. Tremor constant. Cure the rule.

Paralysis Agitans.—Debût often sudden. Tremors incessant. No nystagmus. Tremor begins in the upper limbs, then descends. Locomotion slow at first, then becomes running, as if the patient were always running after his centre of gravity. Intelligence preserved. Very peculiar shape of the hand. No post-mortem appearances known.

(In this our authors are quite mistaken, as reference to Lecture 10 will show.)

Sclerosis Cerebro-Spinal.—Debût generally gradual. Tremor following on paresis. Tremor only on intentional movements. Nystagmus. Tremor first in lower limbs, and ascends. Locomotion uncertain, ‘titabout,’ perhaps from the tremor. Hebetude. Distinct lesions found *post mortem*.

Sclerosis Spinal.—Uncertainty of walk, due to feebleness and paresis of the lower limbs, the initial symptom. Motor troubles due to paresis or paralysis. Locomotion feeble. Vertigo. No loss of equilibrium on shutting the eyes. Sensation normal. Contraction present. Pain in the lower limbs rare. Slight disturbance of sight. Diplopia. Embarrassed speech. Headache. Speech stuttering, never voluble.

Locomotor Ataxy.—Incoördination, preceded by pain in the back, and sometimes by paralysis of some of the nerves of the orbit. No paralysis of limbs, only incoördination. Locomotion disordered. Equilibrium lost if the patient shuts his eyes. Muscles strong. More or less anæsthesia. Peculiar sensation of the soles of the feet, as if the ground were cotton-wool. It is a disease of adult age. Contraction only present in those cases in which the posterior columns have been attacked by sclerosis, and it is a late symptom. Pain in lower limbs usual. Vertigo uncommon. Feebleness of sight, amblyopia, or amaurosis, at first temporary, then permanent; or paralysis of the muscles of the eye, e.g. ptosis, external strabismus at intervals. Dilatation of pupils.

Chorea.—Jactitation, not only of voluntary movements, but often involuntary. Paresis following jactitation. Begins first in the arms, then face, trunk, lower limbs. Walking in irregular jumps. Irritability of temper. A disease of childhood.

General Paralysis.—Arrest of speech in the middle of a word, but the patient is voluble. He has no knowledge of anything being wrong, and it irritates him to tell him of it. Tremor continuous. General paralysis and sclerosis may coexist.

Pathological Anatomy.—1. Lesions visible to the naked eye. Grey circumscribed patches or islets, of greater or less size and depth, expanded without order on the different columns of the cord, or on various regions of the brain. These patches have irregular, but pretty well defined contours. Their dimensions are

very variable, the smallest being almost linear; the largest may measure three to four centimetres in length and two to three in width. Their number is generally pretty considerable.

On touch, they have a consistence firmer than that of surrounding tissue. Sections show that they extend more or less deeply, in the form of nodules or of cones, with badly-defined limits. At the periphery of the cord they are generally rather below the level of cord—sometimes they project. Colour deeper than that of the grey matter of the brain. In chromic acid they are yellowish, then white and opaque, contrasting with the greenish grey colour of healthy nerve substance in chromic acid.

As to distribution, it seldom attacks the grey cortex of the brain. It is sometimes found on the limits of the white and grey, intruding into each. Pretty often it is seen in the walls of the lateral ventricles. It may be in the centrum ovale; in the crura cerebri, looking like secondary degeneration; but in secondary degeneration the lesion has a regular form, and extends from one extremity to the other; whilst in disseminated sclerosis the lesion is irregular, very limited, and coloured like the neighbouring parts. The pons is often attacked by sclerosis. Grey spots, more or less deep, appear on its lower surface. In the medulla oblongata, the olivary bodies are frequently attacked, or one of them; but the pyramids and the other columns may be. In one case the aqueduct of Sylvius was surrounded by a large patch of sclerosis, whence sprang prolongations to the medulla oblongata

and towards the fourth ventricle. The cerebellum is seldom attacked at its circumference. Patches may be visible in its white matter on section, and more especially in the rhomboidal body. In the spinal cord grey spots are seen beneath the pia mater, elongated patches terminating in a point, more symmetrical than in the brain, occupying two opposite columns or several columns on the same side. All the regions of the cord are liable to it. The cranial nerves are usually free; but Cruveilhier found the roots of the hypoglossal, glosso-pharyngeal, and pneumogastric nerves grey; Skoda, the optic nerves hard and flattened; Vulpian, the olfactory and optic, with very apparent patches of sclerosis; Ordenstein, the hypoglossal and motor oculi externus of the left side sclerotic. The spinal nerves are not affected, and may spring quite healthy from the centre of patches of sclerosis. Membranes generally healthy. Muscles often fatty.

(Other observers, however, have sometimes found the spinal nerves sclerotic.)

Microscopically, their observations are very similar to those recorded above; and they come also to similar conclusions—that proliferation of the nuclei and concomitant hyperplasia of the reticulated fibres of the neuroglia are the initial and fundamental facts. Degenerative atrophy of the nerve elements secondary and consecutive.

Whilst these observations are useful up to a certain point, it must be remembered that the aggregate of symptoms does not depend on the special form of

invasion of the disease, but on the foci which are attacked; and when we come to speak of other diseases, you will find there is in many cases no broad line of demarcation between them and cerebro-spinal sclerosis.

Consult—

- Gintrac. *Op. cit.*
 Jaccoud, 'Pathologie Interne.'
 Hammond, 'Dis. of Nervous System.'
 Wilks, 'Path. Anatomy.'
 Rindfleisch, 'Path. Histology.'
 Rokitansky, 'Path. Anatomy.'
 Reynolds, 'System of Medicine.'
 Bucknill and Tuke, 'Psychol. Medicine.'
 Calmeil. *Op. cit.* Vol. ii. pp. 209, 418. 'Softening.'
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 'West Riding Asylum Rep.,' vols. ii. and iii. Dr. Herbert Major.
 'Med. Record,' vol. i. 830. Palmerini.
 'Med. Record,' vol. i. 728. Dr. Batty Tuke.
 Bourneville & Guerard, 'De la Sclérose.'
 'Med. Chir. Rev.,' April 1873.
 'Guy's Hosp. Rep.,' vol. xvii. 185, 190.
 'Med. Chir. Rev.,' July 1874.
 'Med. Record,' vol. ii. 128. Benedikt.
 'Med. Record,' vol. i. 258. Boll.
 'Med. Record,' vol. i. 466. Deiters.

LECTURE V.

TUMOURS.

NOTHING is more difficult than to form a tolerably reasonable classification of tumours of the nervous system. We might take each envelope and each portion of the brain and cord, and mention the tumours that are found in each situation, but such a classification would involve much tautology. Or we might form divisions according to the characters of the tumours themselves, or dependent on the tissues from which they spring. All classifications must be more or less artificial; all are open to objection.

Jaccoud, closely followed by Hammond, divides all tumours of the brain into four varieties:—

1. Vascular, including Aneurisms and Erectile Tumours.
2. Parasitic, including Echinococci and Cysticerci.
3. Diathetic and constitutional, including Cancer, Tubercle, and Syphilitic growths.
4. Accidental and non-vascular, including—
 - i. Fibroplastic, or Sarcomata.
 - ii. Tumeurs a myélocytes.
 - iii. Cholesteoma.
 - iv. Cranial exostoses.

- v. Lipoma.
- vi. Cysts, which may be simple, compound, or containing hair.
- vii. Enchondroma.
- viii. Glioma.
- ix. Glio-Sarcoma.
- x. Cylindroma.
- xi. Myxoma.

He classifies tumours of the cord as—

- 1. Syphilitic, either osseous or periosteal.
- 2. Cancer, including Colloid, Melanic, Sarcoma, and Cysto-Sarcoma.
- 3. Tubercle.
- 4. Fibroma.
- 5. Parasites.

This list is a sufficiently good one, looked at as a mere enumeration, but it is defective as a classification. For instance, aneurisms would come better under lesions of vessels; parasites can hardly be said to form a tumour at all in the brain and cord; to class cancer and tubercle under the head of diathetic tumours, is to make an assertion as to the nature of those lesions that many observers consider as still unproved; whilst it seems scarcely advantageous to make a separate heading of cylindroma, which is an instance of mucous degeneration of a fibroma or a sarcoma.

Gintrac simply enumerates various forms of tumours under the heading of degenerations, and mentions tumours as amyloid, fatty, cholesteatic, and hæmatic (the latter, by the bye, seeming to be merely old extravasations partially reabsorbed and surrounded by a cyst-

wall) lime concretions, bony productions, enchondromata, fibromata, and fibroplastic tumours.

Dr. John Ogle, in 'The British and Foreign Med. Chir. Review,' is not so much attempting a classification of tumours as illustrating certain well-known forms from the resources of two important museums; and with this purpose he speaks of serofulous deposits, purulent deposits, cancer, fibrous and allied forms of tumour, calcareous and osseous deposits, tumours of uncertain nature, and cysts including hydatids.

It seems, however, more reasonable that, as Rindfleisch says, the most important consideration should be, not the individual species of tumours, but their seat, the tissue from which they proceed.

He divides tumours into three varieties; viz. :—

1. Tumours which proceed from the free surfaces of the envelopes and interior cavities of the nervous system.

2. Tumours which proceed from the sheaths of the vessels.

3. Tumours which have their origin from the neuroglia.

Under the first heading, tumours which proceed from the free surfaces of the envelopes and interior cavities of the nervous system, he classes :

- i. Pacchionian granulations.
- ii. Spindle-celled Sarcoma.
- iii. Myxoma.
- iv. Psammoma.
- v. Lipoma.

Under the second heading, tumours which proceed from the sheaths of the vessels, he classes :

- i. Carcinoma cerebri simplex.
- ii. Fungus of the dura mater.
- iii. Cholesteoma.
- iv. Epithelioma myxomatodes psammosum.
- v. Papilloma of pia mater and vessels.
- vi. Papilloma myxomatodes.
- vii. Gumma syphiliticum.

Under the third heading, tumours which have their origin in the neuroglia, he places :

- i. Glioma.
- ii. Myxoma of the nerve substance.
- iii. Tubercle.
- iv. Fibroma, including false neuroma.

Following this classification as convenient, though by no means perfect, let us see where these various tumours are situated, and of what elements they are composed. Taking them, then, in the order above mentioned :

i. Pacchionian granulations of the arachnoid, especially along the longitudinal sinus ; non-vascular groups of papillæ, consisting of striped connective tissue, poor in cells, proceeding directly from a thin but a continually renewing layer of sub-epithelial germinal tissue.

ii. Sarcoma. Less frequent than cancer ; these productions have a variable volume, seldom attaining the size of an apple. Exceptionally they may be developed in the thickness of the nervous tissue, but they generally spring from the dura mater, and more often at the base than the convexity. Sarcoma may also spring from the dura mater spinalis. Rokitansky

has seen some that had originated from the pia mater or the ependyma, but this starting-point is rare. Their consistence varies. In some cases it is nearly allied to that of fluid tumours; in others the mass is soft; it presents a sort of gelatinous transformation; sometimes it is pitted with small cavities, filled with a clear, yellowish, non-viscous fluid. These tumours are composed of fusiform bodies, nuclei, and vessels; amorphous matter is also met with, connective tissue, and less frequently fat vesicles.

iii. Myxoma, generally from the convexity of the brain; the brain accommodates itself to its presence easily. Also from the dura mater spinalis, and here presses dangerously upon the spinal cord.

Jaccoud speaks of these tumours as having their origin from the spheno-occipital synostosis; as being very small, attaining the size of a filbert at most, soft and fragile, having a gelatinous aspect. The small mass is attached by a pedicle to a small cartilaginous excrescence springing from the spheno-occipital suture, appearing free between the dura mater and the arachnoid, or adherent with the arachnoid to the centre of the pons. These tumours are rare.

iv. Psammoma. A tumour with a connective tissue, or even with a mucoid tissue substratum, which is distinguished by its contents of globular lime concretion, as in the pineal gland. These growths have been met with at once in the brain, spinal cord, spinal membranes, and nerves. They are not uncommon in the choroid plexus. They vary in size from a pea to a nut. It is probable that these tumours have always

a connective tissue origin, and that when they are found with a mucoid tissue substratum they are really examples of mucoid degeneration.

v. Lipoma. Rare. Small lipomata may be found springing from the inner surface of the dura mater cerebri, and from the ependyma of the ventricles. Dr. Coats of Glasgow has published a case of a fatty growth connected with the pia mater over the corpus callosum. Gintrac speaks of lipoma as a disease generally of foetal or very early life, and especially of the ventricular walls. Later on they may attack the grey matter of the convolutions and the corpora striata, and in adult age the meninges. The fatty matter is often contained in a kind of cell, these cells being surrounded by a common membrane or cyst, and provided with vessels.

The second heading comprises :

i. Carcinoma cerebri simplex. Most frequent from the under surface of the pia mater. The first link of this process is the formation of larger cell-heaps out of each adventitial cell. The cells at the centre of the ball are globular, at the surface spindle-formed. The spindles of adjacent cell-heaps come together with incomplete fibrous lines, and thus form finer septa, which subdivide the larger alveoli of every carcinoma. In the further increase of the tumour the cell proliferation predominates. The vessels from which the new formation proceeds are in a great measure obliterated to thin lines of connective tissue. This is Rindfleisch's description.

Cancer may spring from the bones, the meninges,

the orbital cavity, or the brain : when the brain is the seat of origin, cancer may attack, in order of frequency, the cerebral hemispheres, the cerebellum, the optic thalami, the corpora striata, and the pons. Less frequently are the corpora quadrigemina, the fornix, and the medulla oblongata attacked. Cancer of the orbit or of the cranial bones may proceed inwards, and cancer of the brain outwards. The size of the tumours is very variable ; the largest are those which proceed outwards, or which occupy the cerebral hemispheres.

When cancer is primary, which in the brain is generally the case, it is mostly single ; if it is secondary, or if it coincide with cancer of viscera, it is often multiple, and then may be perfectly symmetrical.

Unless it proceeds externally, cerebral cancer undergoes no ulceration or sanious softening ; indeed it may partially retrograde, its elements becoming atrophied or fatty, and the tissue is transformed into a compact homogeneous caseous mass, in which the vessels are destroyed. The stroma may be incrustated with lime deposits.

The principal varieties of cancer are encephaloid, much the most common, then scirrhus, and colloid. Ogle gives ten instances of cancer springing from the external cerebral membranes, two from the ependyma, twenty-two occupying some part of the cerebrum, three having their origin in the bones of the skull, two in the cerebellum, and two in the medulla oblongata.

ii. Fungus of the dura mater. It arises from the side of the dura mater turned to the bone, penetrates

with the vessel into the compact substance, destroys the vitreous table, thereupon spreads out somewhat more easily in the diploe ; finally breaks the external compact lamella of the cranial bones to lift up the integuments of the cranium as a fungoid proliferation.

In the catalogue of the Pathological Department of the Oxford Museum, Van der Kolk makes some interesting remarks with reference to specimen 803, three so-called fungi of the dura mater. These tumours were supposed by Wenzel to spring from the dura mater ; by Walther, from the diploe of the cranium ; by Chelius, sometimes from the dura mater, sometimes from the diploe, sometimes from the periosteum. To settle the point, he says, I injected the middle meningeal artery on either side, and found that the injection passed freely into the fungi, thus showing that they grew from the dura mater itself. But the outer of the layers of which the dura mater is composed is nothing but periosteum, and therefore these fungi are diseases of the periosteum.

iii. Cholesteoma. Pearl cancer. It unites the structure of an epithelial carcinoma with the innocuity of a wart. It is a pavement epithelium, whose cell cylinders have been entirely converted into a mass of pearly globules, shining like satin, most frequent at the base of the brain, and occasionally as large as a walnut. It is covered upon its surface by the arachnoid, and evidently springs from the vessels of the pia mater. Sometimes it is found in the centre of the brain substance, having its origin in the lymph spaces and sheaths of vessels. Some observers say that

a tumour of this kind may contain a good deal of cholesterine and stearine. It may be connected with the meninges, the brain, the cerebellum, and the spinal cord. Its form is irregular, with an envelope of a certain fibrillar arrangement, and with the parenchyma of the tumour disposed in connective layers; both envelope and parenchyma are nearly non-vascular.

iv. Epithelioma myxomatodes psammosum. It is very doubtful whether it is scientific to draw any line of demarcation between this tumour and the preceding. It is closely allied to it. It consists of epithelial cell-cylinders and nodules imbedded in a very voluminous stroma of mucoid tissue. It seems really to be an instance of mucoid degeneration of pearl cancer, with the addition of lime concretions. It is rare. It has been met with in the third ventricle.

Perhaps this form and the two following might be included under the cylindroma of Jaccoud.

On looking back upon a case I saw a good many years ago, I believe it was an instance of this disease:

Man, aged 52. Calvaria very dense. Dura mater adherent along the superior longitudinal sinus. Brain itself rather soft, especially the crura cerebri. Cerebellum very soft. Pituitary gland very prominent. Beneath it and all round, the dura mater was detached from the bone, and there was a soft feeling beneath it. On section this was found to depend upon a mass of soft colloid-looking material, which was united to the pituitary gland, and occupied the site of nearly all the body of the sphenoid bone, which it had destroyed, part also of the ethmoid, and the basis of each orbit,

with the nerves entering each orbit. The colloid mass was the seat of small hæmorrhages. Under the microscope it was composed of granular cells of very various shapes. The disease had attacked the dura mater lying over it.

v. Papilloma of the pia mater and vessels. This tumour is reddish-grey, translucent, tremblingly soft. It breaks up into a mass of reddish branched papillæ, each of which has a central vessel, very little connective tissue, and a doubly stratified epithelial mantle. The stroma of the papillæ is connected with the blood-vessels.

vi. Papilloma myxomatodes mostly occurs multiple in the brain. It is distinguished from simple papilloma by the abundant production of mucus at the surface of the papillæ. The epithelium consists of very large well-formed cylindrical cells, which secrete the mucus.

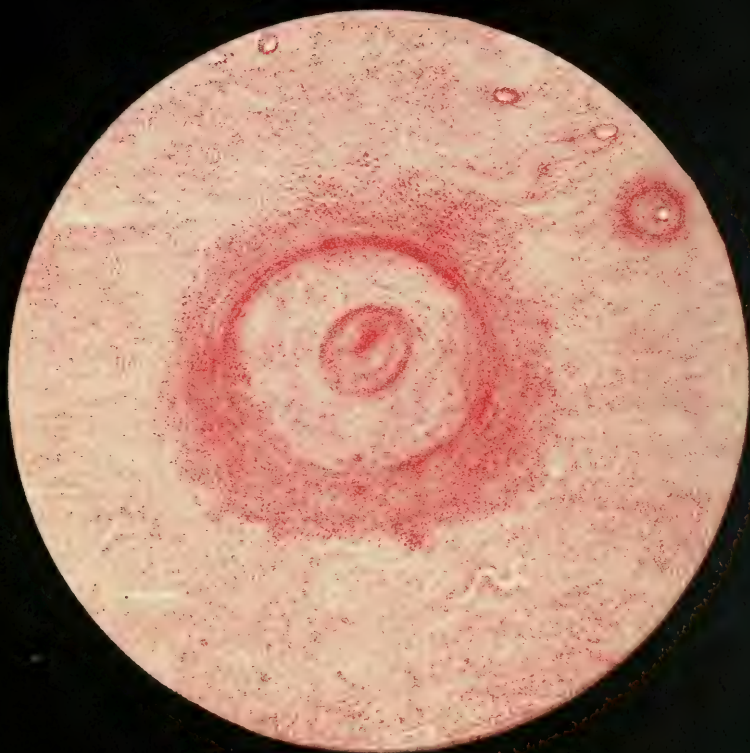
vii. Syphiloma of the nervous centres. Syphiloma of the nervous centres exists sometimes in the state of diffuse infiltration, but most generally it forms a circumscribed swelling, a true tumour. It is situated in the meninges alone, or rather in these membranes and in the cortex; it rarely occupies the nervous tissue alone. Syphiloma is never encysted, and even when it appears to constitute a well-limited tumour, it is really diffused at the circumference, and penetrates for a certain distance into the normal tissue; the volume is very variable, but in the brain it never attains the considerable dimensions that it sometimes presents in the lung or the liver. The tumour appears as a soft homogeneous mass of reddish-grey, sprinkled with

bloody puncta in certain cases ; this substance furnishes no juice, or rather it gives out a juice limpid and opalescent, and, as it were, mucous. These productions are essentially composed of nuclei and cells ; these latter occupy sometimes the centre of the mass, whilst the nuclei are at the circumference ; the nuclei, which generally contain a very visible nucleolus, present besides nothing particular ; the cells are like the white globules of the blood with a single nucleus ; these elements, with which we sometimes find some bodies of a doubtful cellular nature (the protoplasms of Wagner), occupy the meshes of a connective network of new formation, and from the reciprocal relations of the cells and the stroma there results a finely alveolar texture.

The most frequent further transformation is atrophy of the cells and of the nuclei, either simple atrophy, or combined with a fatty degeneration of greater or less extent.

The point of origin of syphiloma is generally the normal connective tissue ; but for the brain and all the organs poor in tissue of this kind Wagner is led to admit that the cells and the nuclei proceed from the multiplication of the nuclei of the capillaries, and that the stroma is formed by the further fibrous metamorphosis of the wall of the vessel. This process is not constant, and in a good number of cases there is really a new formation of connective tissue.

Dr. Broadbent has lately given an exhaustive account of syphilitic lesions of the nervous centres. He says that, generally, a small part of the organ is attacked, and the remainder is left quite free. The



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disease is strictly localised in the spot it affects. The outer part is composed of fibrous tissue, which can be seen to represent the natural fibrous supporting elements of the part in a state of augmentation, while the functioning elements of the part have dwindled away. It is a local sclerosis. See plate 11, in which this fibrous condition is beautifully seen in a concentric arrangement. Dr. Moxon's plate of syphilis of the spinal cord in 'Guy's Hosp. Reports,' vol. xvi., is somewhat similar.

The central part of the syphilitic nodule shows the caseous or gummatous faint yellowish matter of more and more elastic consistence, and less and less friability and curdiness, generally rather sharply distinguished from the fibrous outer part, and sometimes softening down or calcifying. There are signs of more acute inflammation in the immediate neighbourhood, showing lymph or adhesions to the part around.

Dr. Moxon says that syphilis attacks the surface of the brain and its membranes; it attacks them in limited spots, and it spreads slowly. The morbid changes are, on the one hand, adhesions of the membranes to each other and to the surface of the brain, by means of an adventitious material of firm consistence and yellow colour, which may be called lymph, but is harder, tougher, and more opaque. This exudation may be found at any part of the surface; it invades and destroys the grey matter, interferes with the supply of blood, and, when it occupies the membranes at the base of the brain, surrounds and involves the nerves in the intracranial part of their course.

Locomotor ataxy is not directly caused by syphilis;

but it is easily conceivable that in multiple syphilitic deposits in the cord a sufficient number might be situated in the course of the posterior white columns and grey matter to impair spinal coördination. Acute general or local myelitis may be caused by syphilis, especially in the secondary stage, inflammatory softening being set up round a small gumma. Spinal meningitis may also be due to syphilis. Small syphilitic tumours may exist both in the pons and the medulla longata, and the cerebellum may probably be affected in a similar manner. The habit of locality of the tumours, as well as of the diffused exudations, is to affect the surface, although gummata may be found in the substance of the brain; usually, however, in the more vascular parts, the grey matter of the corpora striata or thalami. In the diffuse form we may have the membranes adherent to each other and to the convolutions by means of firm plastic material; and, as a result, the vessels of the pia mater are occluded, the supply of blood to the peripheral grey matter is diminished, and this undergoes atrophic change of some kind; or small indurations may invade the nervous structures and the membranes.

The following case showed some of the usual appearances:—Woman, aged 50. The dura mater rather roughened on its inner surface, over the space of a five-shilling piece, on the upper surface of the middle lobe of the right hemisphere. At this spot all the membranes were adherent to the brain. At this spot also the brain itself was very hard, but smooth, and there was no appearance of convolution here. On

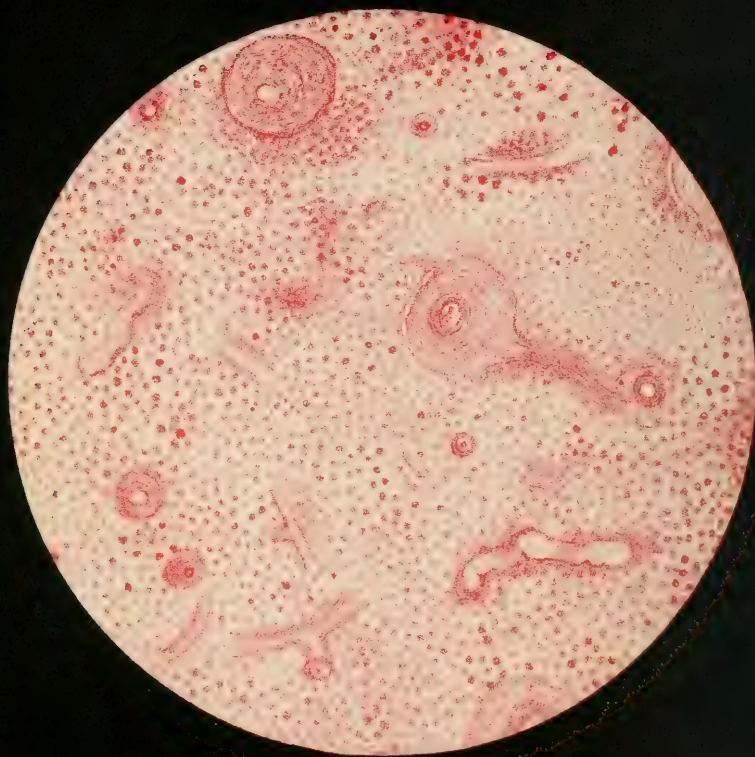
section vertically, a small tumour was cut through, hard and yellow, like those of syphilitic origin. It was the size of a small walnut. The grey matter immediately around it was indurated; all the rest of the grey matter was healthy. The white matter was universally soft in this hemisphere, almost diffuent in some places, and totally without bloody puncta. The septum was entirely gone, and only the fine membrane left. No fluid in the ventricles. Arteries healthy. Left hemisphere healthy.

Convulsions depending upon epilepsy may be produced by tumours growing either from the bones or dura mater, or in the pia mater, or in the substance of the hemispheres, where the tumours reach the surface; or by diffused exudation in the pia mater, or by thrombosis at the time of its occurrence and during the consecutive changes; or by slighter changes affecting the nutrition of the hemispherical grey matter.

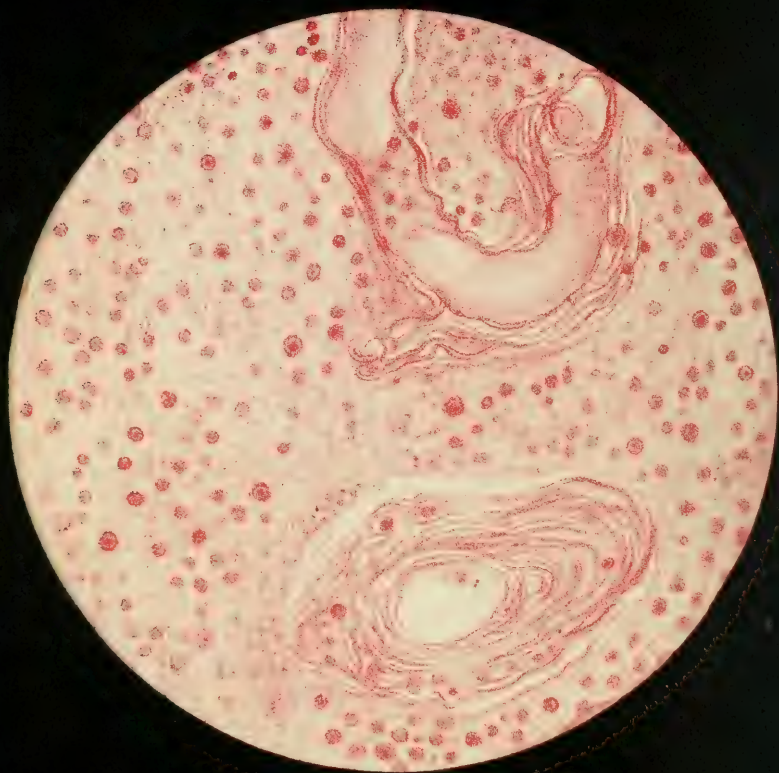
Speaking generally, and still drawing much from Dr. Broadbent's experience, paralysis of cranial nerves and of the limbs, gradual in its mode of access, is characteristic of disease about the base of the brain; convulsions and mental affections of disease of the convex surface of the hemispheres. No strict line of demarcation can be drawn between cases in which there is extensive exudation in the membranes and those in which the morbid process results in the formation of distinct tumours. In the former, the deposit frequently here and there takes the form of a nodule, which projects into the brain substance; and a syphiloma is accompanied by or sets up changes

in the adjacent part of the meninges. Gros and Lanceraux give one case in which the entire mass was infiltrated; in another, disseminated exudations existed in different parts; in six, tumours were found in the anterior lobes; in three, in the middle lobes; in three, in the corpora striata; in three, a great part of the hemisphere was involved.

Double optis neuritis is said by some observers to be an important symptom of these and other cerebral tumours. I have no doubt that the ophthalmoscope gives important aid in determining the condition of syphilitic meningitis or of syphilitic tumour. If the latter is present, and is not complicated with meningitis, its effect on the retina is to produce choked disc, as the result of pressure interfering with the venous reflux from the retina. On the other hand, if basilar meningitis of the anterior lobes is present, we may have true optis neuritis from the spreading downward along the nerve of the meningeal inflammation. This is usually a gradual process, and more likely to follow syphilitic than any other variety of meningitis. The real difficulty lies in the fact that syphilitic tumour is not unfrequently associated with a certain amount of meningitis. Cerebral disease in infantile syphilis may be mistaken for tuberculous meningitis. In Dr. Broadbent's case there was lesion of the left occipital lobe, which was hard and shrunken. The induration involved the cuneiform lobule. The pia mater was adherent and almost black. The grey substance was atrophied; and the white, firm and tough, like leather. The posterior corner of the lateral ventricle was



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inclosed by this indurated white substance. There was also superficial induration of the posterior part of the left optic thalamus, and some induration of the superficial transverse fibres of the pons.

The arteries of the brain in syphilis are frequently attacked with inflammation, usually beginning in the outer coat. This may lead to thrombosis, which cuts off the supply of blood, and produces the results now known to follow this event. The effects are first an accumulation and stagnation of blood in the capillaries in the area of distribution of the vessel blocked; and unless collateral circulation can be established, there will be subsequent softening.

The Third Form.—Tumours which proceed from the neuroglia. The cells of the neuroglia are the starting-point.

i. Glioma. An abnormal development of connective tissue, more usually found in the posterior cerebral lobes. It may be as large as an orange. It is of two kinds—one soft, of the consistence of brain substance; the other much harder. It consists of cells and nuclei, but never contains any of the nervous elements. It is practically a neoplasia of the neuroglia; and, on minute examination, is seen to consist of cells of connective tissue, of vessels, and of cells resembling amyloid bodies, but which give rise to no iodine reaction.

Plate 12 is an example of glioma of the brain.

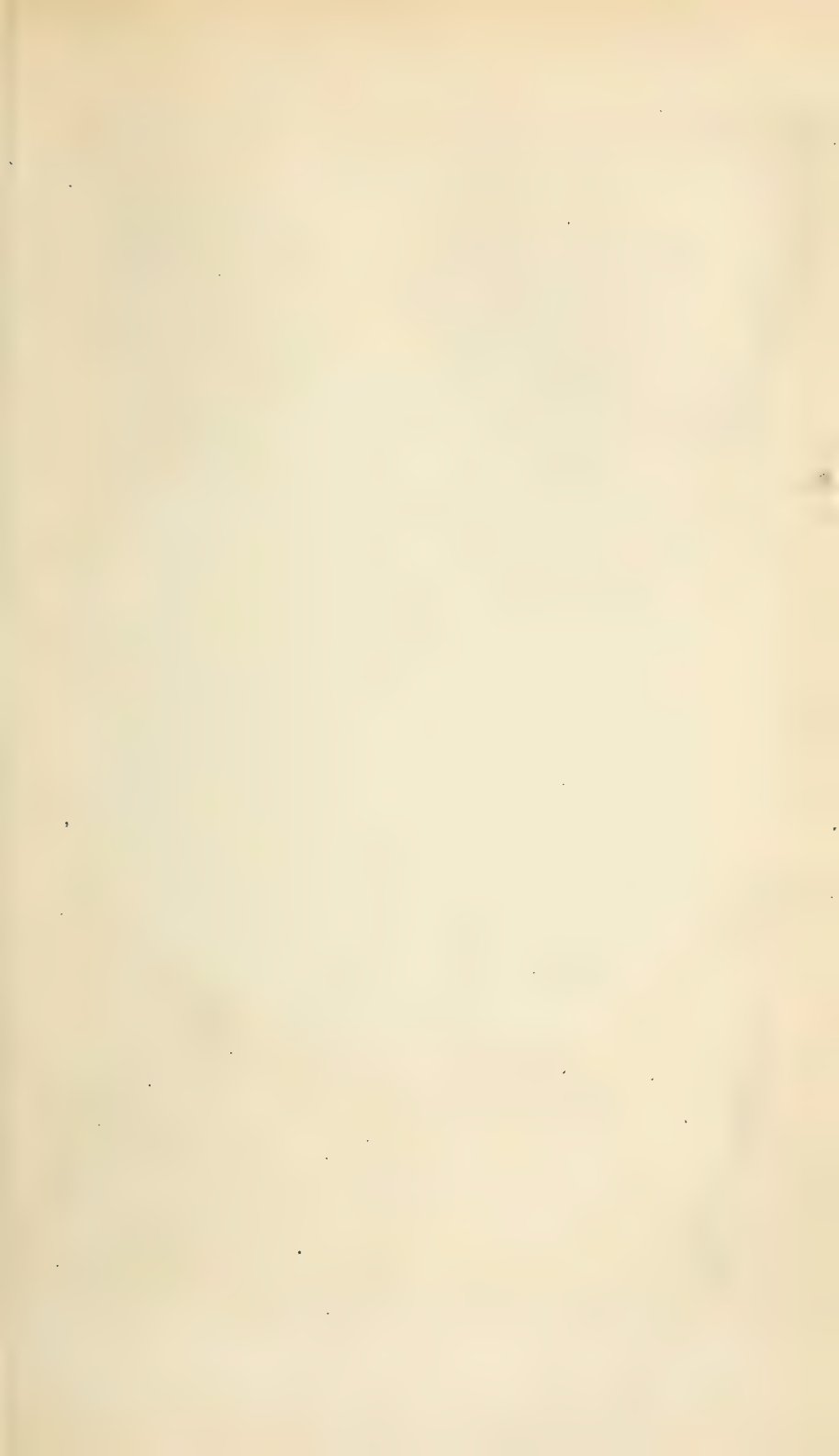
Plate 13 is a second specimen of the same, showing that the vessels are much implicated, being specially thickened.

Dr. S. Smith's account of the microscopical appearance of the vessels in his case, reported in the 'British Medical Journal,' June 6, 1874, is, that in the membrane surrounding the tumour, but not in the substance of the tumour itself, the vessels were all surrounded by a cellular infiltration of the intervening tissues, and the perivascular lymphatic sheaths were much distended by epithelial proliferation from their walls: the calibre of the vessels was much diminished by the surrounding cell-growth. Small masses of red corpuscles were seen, surrounded by a cell-growth, extending to as much as twelve times the diameter of the calibre of the vessels, the growth being of a fibronuclear structure, arranged in a concentric manner around the calibre of the vessels, and limited externally by a finely fibrous investing membrane, no muscular coat being visible.

ii. Myxoma of the nerve substance. More common in the spinal cord than in the brain, and more common on the peripheral nerves than in the spinal cord. The myxoma proceeds from a hyperplasia of the nerve-connective substance, and uniformly extends by infiltration in all directions. The fibres of the spinal cord or of the peripheral nerves are crowded asunder and partially perish. On the nerves myxoma presents itself as a spindle-formed tumefaction.

It forms a variety of neuroma.

Genuine neuroma contains pre-eminently newly-formed nerve-fibres. Another form of neuroma possesses no new nerve-fibres; it is really a fibroma in the substance of a nerve, and is produced by a local new formation from the interstitial connective tissue;





FIBROMA OF 4TH CERVICAL NERVE

this interstitial proliferation has most frequently the fibroid character, more rarely that of myxoma, or the softer forms of lipomatous sarcoma.

Plate 14 is an example of the fibroid form of false neuroma, from the Oxford Museum.

The specimen was from a girl, aged 24, who had long complained of pain in the left scapula and shoulder. To this succeeded numbness of the left arm and foot, and then paralysis of these parts, with violent attacks of pain and tetanic spasms in all the limbs.

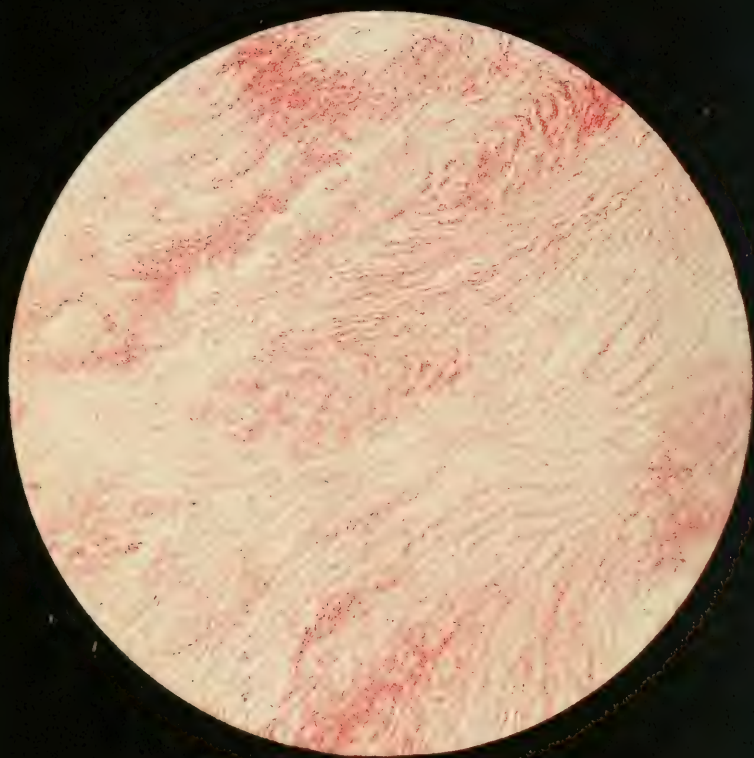
The neuroma is covered by a thick fibrous capsule; it has stretched and elongated in a remarkable manner the root of the fourth nerve, so that the posterior root of the nerve seems to have been injured by it; it is contained or shut in the sac of the dura mater. At the posterior surface of the medulla are abundant false membranes, which have glued together the medulla and the dura mater.

iii. Tubercle. Tubercle prefers the hemispheres and the cerebellum; it is seldom found in the optic thalami and corpora striata, and is only exceptionally met with in the pons and the medulla oblongata.

Ogle has found a scrofulous mass on the dura mater, covering the right lobe of the cerebellum, and implicating the brain at the same time; twice the tentorium cerebelli was affected; twenty-three times the masses were connected with the pia mater, of which in two cases the choroid plexus was affected; in one the velum interpositum, and in one the ependyma of the ventricles was the seat; twenty-seven

times the tuberculous masses were found in the substance of the cerebrum itself; twenty-eight times in the cerebellum; once in the left crus cerebelli; four times in the medulla oblongata, in one of which a similar mass was found in the substance of the sixth cranial nerve; and five times in the pons. It is scarcely necessary to say that in many of these cases the scrofulous masses were multiple, and existed in various organs of the encephalon at one and the same time.

Gintrac has found tubercle in all portions of the meninges, in the brain, especially in the cortical substance, in several situations at once, in the cortical substance only, in the middle lobes, in the anterior and the posterior, in the optic thalami, generally solitary in this situation, and in the pituitary gland; in the cerebellum, its right lobe, its left, and both lateral lobes, in the central region of the cerebellum alone, and in this situation as well as the lateral region. He finds it generally solitary in the mesocephale, especially in the crura cerebri; but it may coexist in the mesocephale and the brain; in the mesocephale and the cerebellum; in the mesocephale, the cerebellum, and the brain; and the mesocephale, the cerebellum, the brain, and the meninges. It is also generally solitary in the medulla oblongata, but has been found coincidentally in the medulla oblongata and the brain, the medulla oblongata and the cerebellum, and the medulla oblongata and the mesocephale; whilst the same rule obtains in tubercle of the spinal cord, although it may exist at once in the spinal cord and the brain.



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Rindfleisch considers that the so-called tubercle of the brain occurs under two forms; one a fibroma, non-tuberculous, in which it is seen that the number of cellular elements in the nervous substance always increases the nearer we approach the zone of proliferation, and that a layer of round-celled germinal tissue is at the same time the matrix of the tumour and the product of the cerebral substance, and that there is a transformation of the germinal tissue into fibres, and that there are no shrivelled cells.

The second form, the caseous nodule, often multiple and smaller, not homogeneous, but composed of round portions, will agree in form and size with miliary tubercle. The caseous nodule contains dead, the grey proliferating zone still living, miliary tubercles.

This seems to me to be the weakest point in Rindfleisch's classification. Whatever may be true as to the starting-point of these small fibromata, the seat of origin of tubercle in the nervous centres is probably always, or at least most generally, the vessels. What can be seen to be the case in the minutest point of tubercle is most probably the case in the caseous nodule, which is simply an agglomeration of miliary tubercles.

Plate 10 is an example of a small fibroid tumour, non-tuberculous, implicating the spinal cord.

I much prefer Jaccoud's account of these tubercles. They occupy the white and the grey substance equally, and present themselves under the form of small isolated circumscribed masses, varying in number from one to

twenty, and seldom exceeding the latter. Their volume is in inverse ratio to their number. Pretty often they are the size of a cherry ; at other times they scarcely exceed the size of a grain of wheat. As to the colossal masses, which attain to the magnitude of a hen's egg, they result from the confluence and fusion of several spots originally distinct.

These tumours are of a greenish-yellow colour. The small tumour is only a mass of caseous deposits, which are most frequently encysted by a layer of connective tissue, which quite separates them from the nervous tissue.

According to the observations of Cornil, these yellow nodosities are generally made up of two very distinct parts, which appear on their surface on section ; one central, opaque, and yellow, dense, hard, dry, deprived of juices and of vessels visible to the naked eye ; the other, peripheral, semi-transparent, less dense, more succulent, and allowing one to see with the naked eye some few red lines indicating vessels. At the point where the vessel penetrates into this semi-transparent zone, the aspect of the elements which surround it changes completely ; the small cells or the nuclei, agglutinated one to the other by a granular mass, fill the space between the lymphatic sheath and the muscular layer, and the cavity of the vessel, instead of being permeable and free, is filled with coagulated fibrine. The coagulation and impermeability are absolutely constant in the opaque and yellow central mass of these tumours. This character distinguishes tubercles from the 'tumeurs à myelocytes' of Robin, in which the vessels remain permeable. These deposits are sometimes encrusted

with salts of lime, but they have little tendency to diffuent softening and suppuration.

Féréol, *Med. Record*, v. 1, 566, reports a case of confluent tubercle of the pons varolii just at the nucleus of origin of the left sixth nerve, the phenomena having been paralysis of the left sixth nerve, and conjugate inaction of the internal rectus of the right eye.

Syphilitic tumours may become caseous, and must be distinguished from tubercle.

This classification of Rindfleisch takes no direct notice of Cysts of the nervous centres. A proliferation of the connective tissue round hæmorrhages, collections of pus, and various growths, cannot be spoken of under the name of tumour. But other cysts are met with of more or less independent origin; cysts, for instance, from the internal surface of which hair has been secreted, or thick honey-like fluid, or fibrine. It is not certain that the two latter varieties may not have been formed originally round a hæmorrhage; but the cysts containing hair are certainly of a different nature. They are very rare. Serous cysts, moreover, are met with connected vascularly with the neighbouring parts. They are attached to the cranial and spinal meninges, especially the pia mater; in the cerebral ventricles, especially attached to the choroid plexus; they are met with in the brain, cerebellum, and mesocephale. They are generally solitary. They are composed of two or three layers of serous, fibrous, or cartilaginous tissue, and are made up of connective tissue, fibrous and fibro-plastic elements, and contain in their structure or on their internal surface amyloid bodies, cholesterine and crystals

of triple phosphate. The interior may be lined with an epithelial expansion. For arachnoid cysts see Lecture 7.

Dr. Ogle speaks of five cases he has found in the museums of cysts connected with the dura mater, nine with the pia mater, thirty-two occupying some part of the brain, of which one was connected with the pineal gland; two of these, however, were most probably hydatid cysts; in five the cyst was situated in the cerebellum, in one in the medulla oblongata, and in one in the pons.

Parasites.—Hydatids occur in the nervous centres in various situations.

- a.* Developed outside the meninges in connection with the cranial bones.
- b.* In the meninges.
- c.* In the brain; in the cerebral substance; in ventricles and the choroid plexus.
- d.* In the spinal canal, generally attached to the meninges.

Cysticerci.—More rare; not found connected with the cranial bones, nor in the spinal canal.

- a.* Developed in the meninges of the brain.
- b.* In the substance of the brain and cerebellum.
- c.* In the ventricles and the choroid plexus.

Von Gräfe once observed a Cœnurus in the encephalon.

Consult—

Hammond, 'Dis. of Nervous System.'

Jaccoud, 'Path. Interne.'

Wilks, 'Path. Anatomy.'

Gintrac. *Op. cit.*

Rindfleisch, 'Path. Histology.'

Ogle, 'Brit. and For. Med. Chir. Review,' 68, 70, 71, 72.

Broadbent, 'Brit. Med. Jour.,' 74, vol. i.

'Guy's Hosp. Rep.,' vol. xvii. 'Syph. of Brain.' Dr. Moxon.

'Guy's Hosp. Rep.,' vol. xvi. 'Syph. of Cord.' Dr. Moxon.

'Guy's Hosp. Rep.,' vol. xvii. 'Strumous Tumours.' Dr. Habershon.

Lanceraux, 'Treatise on Syphilis.'

'Jour. of Mental Science,' April 73 and Jan. 73. Dr. Boyd.

'Jour. of Mental Science,' July 72. Dr. Clouston.

'St. George's Hosp. Rep.,' vol. iv. Dr. Allbutt.

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LECTURE VI.

DELIRIUM.

ACUTE DELIRIUM. Postponing for the moment the consideration of *délire aiguë* of French authors, acute delirium is met with under very varied conditions of blood supply. It is not by any means associated in all cases with hyperæmia of the brain. On the contrary, all medical men will recognise in their experience the truth of Dr. Bucknill's statement, that the fact that all the symptoms of acute mania frequently arise and continue throughout the course of an exhausting bodily disease, which leaves every individual organ, the brain included, in an ill-nourished and anæmic state, affords irresistible evidence that the phenomena of acute insanity must in many cases coexist with a state of the cerebral organs the very reverse of hyperæmia. Graves records a case where a patient, though violently delirious and unmanageable, on the borders of frantic madness, had a pulse almost uncountable from quickness, and exceedingly weak, with cold extremities. Closely connected with this point is the condition of the urine in acute delirium. If there were a constant hyper-secretion of phosphates in all cases of acute delirium, we should be nearer a reasonable explanation.

Acute delirium, in fact, would be a symptom of excessive brain action. But this is far from being the case. In one form of the disease, delirium tremens, in which at times the delirium is most acute and violent, the phosphates are generally diminished very considerably; and it is not even proved satisfactorily that acute delirium, with manifest hyperæmia of brain, is in all cases accompanied or followed by abnormal increase of phosphoric acid in the urine. This anæmic acute delirium is seen sometimes at the end of exhausting diseases, or after severe hæmorrhage, as flooding after parturition. A condition not dissimilar is seen in superlactation; or, again, some days after the birth of a child acute delirium may supervene, and may have its origin either from exhaustion or from septicæmia. The thermometer here is a great aid.

Next to the anæmic form we meet with acute delirium depending upon toxæmia. We see this condition from the exhibition of certain substances, such as alcohol, sulphuric ether, belladonna, datura stramonium, &c. It is this form that we find at the commencement of acute diseases, inflammation of any kind, fevers, &c. This form obtains also in rheumatic pericarditis, although it is thought by some that this delirium was an anæmic kind, due to the interference with the heart's action. It is worthy of note that true rheumatic meningitis is generally accompanied by a low muttering delirium. It is this form that we meet with at the close of phthisis, due to the circulation in the brain of morbid blood. In some few cases the presence of an abnormal amount of carbonic acid in the blood gives rise to

acute delirium. This seems to be the rationale of the mental phenomena of delirium tremens, and it is sometimes seen in fatal cases of pneumonia. In erysipelas the morbid blood is again the cause of the delirium. It is a difficult question to decide whether the delirium is caused in these diseases by a blood rendered impure, or by the heat of the blood. At any rate, it is remarkable how anything that reduces the heat of the blood seems to relieve the delirium.

It is not easy to say what is meant by excitability of brain, apart from the calibre of vessels. Let it be granted that there is no abnormal increase or deficiency in the amount of blood in the brain, and that the blood is not morbid in quality, can any condition of brain exist that will give rise to acute delirium? No doubt this state of delirium may come on after shock of any kind, but it is doubtful whether shock can affect the brain except through its vessels.

The whole subject is full of difficulty. The use of the word 'polarity,' as applied to the brain cells, is no real explanation of any known condition. It is a mere hypothesis from analogy. It may be true that the brain is made up of a series of batteries, but it is far from being a proved fact. On the one hand, the known convulsive phenomena and loss of consciousness consequent on arterial spasm, the fact of the regions of epileptic discharge being governed by the distribution of vessels, and the morbid cerebral phenomena occasionally met with immediately after an epileptic fit; and on the other hand, the lesions found in the cells themselves in all cases of prolonged abnormal

cerebral phenomena, such lesions being constantly due to previous alterations of blood supply, make it difficult to realise the existence of acute delirium, the cells being healthy, except under one or other abnormality of vascularity, expressed by the varieties of anæmia, toxæmia, or hyperæmia. So far we see anæmia, toxæmia, heat of blood, and perhaps shock, as conditions under which acute delirium may be a prominent phenomenon. Dr. Handfield Jones considers that in many instances the excitation of the tissue is the primary change, and the vascular repletion is secondary and varying.

My own belief is that delirium is in all cases an expression of functional inactivity or perverted activity, accompanied or caused by deficient blood supply; in anæmia from deficient circulation; in toxæmia from the circulation of the blood, part of which, at least, is unfit for functional purposes, and therefore useless; in hyperæmia, because from the very pressure upon the vessels the due interchange between tissue and nutritive material is rendered abnormally difficult.

It stands to reason, therefore, that, except in the latter condition, the post-mortem appearances will be few. The brain may be abnormally pale in anæmia, and if it has been of long duration, we may find either slight partial softenings, or slight atrophy of brain and œdema of the pia mater, or at least very empty vessels. But on this point it must be remembered that, owing to the facility with which the cerebral vessels empty themselves, considerable hyperæmia of brain may have existed and yet leave no trace behind, unless it has

been very prolonged. In toxæmia there may be spots of local congestion or of pigmentation, but in most cases the post-mortem appearances will be simply none.

In hyperæmia, however, the case is different. It may be we find a brain abnormally red, and showing on section many puncta vasculosa. Or part of the brain may exhibit the cribriform condition already alluded to, due to the pressure the tissue has undergone from dilated vessels; or the vessels themselves may be dilated, as in some cases of epilepsy, and a vast amount of hyperæmia of the brain and so-called congestion is subsequent to an epileptic attack; or, if the hyperæmia has been of long duration, the muscular coat of the vessels becomes hypertrophied.

In other cases a pigmental condition of the walls of the vessels is observed, and the smaller arteries will be found aneurismal or tortuous, or here and there permanently dilated. In some cases the altered vessels will have given way, and the results of the hæmorrhage (crystalline hæmatoidine, &c.) may be recognised.

Acute delirium is a common symptom of acute meningitis, though probably not of the meningeal lesion proper. Meningitis is so intimately allied with lesion of the brain, so much so, indeed, as to induce Trousseau to call this malady by the name of cerebral fever, that it would be difficult to say which symptoms are caused by the inflammation of the meninges, which by the inflammation of the brain. Most probably these phenomena are directly associated with the hyperæmia of the convolutions so constantly found in this malady.

In such cases there will be all the special lesions of meningitis.

In many cases the cerebral congestion is of temporary duration, and does not lead to inflammatory lesion.

In some few cases the extreme hyperæmia has seemed to have been the commencement of local spots of encephalitis. Calmeil gives many such cases in his first volume, and it is likely that it is always a very early phenomenon in such cases, though it may not be the primary one.

Acute delirium may accompany periencephalitis in insidious forms. Calmeil gives 19 cases in which this was the case. In 9 the tissue of the cranial bones was notably coloured red. In 8 the vessels of the cerebral dura mater were red and arborescent. In 2 cases the cavities of the cerebral arachnoid contained a certain amount of purulent liquid; in 3 of serous.

In 4 the cerebral pia mater was infiltrated with serum; or with a liquid of a plastic appearance in 8 cases, with pus in two.

In 3 it was separated with difficulty from the nervous substance beneath it. In 7 it adhered intimately in spots to this substance.

In 18 its folds were red, its network more or less rugose by injected vessels, sometimes of considerable calibre.

In 18 effusions of blood had occurred in its thickness.

In 7 the convolutions of one hemisphere, or both, or of certain spots of the brain were swollen, and more or less augmented.

In 1 they were tinted brown by contact with pus.

In 18 the cortex was red, rose, or violet colour, either externally or in its thickness; in 9 it was of too little consistence; in 1 it contained a small centre of tubercle; in 1 a small centre filled with cholesterine.

In 14 the vessels of the white matter were injected; in 4 this substance was loose and of a white consistence; in 1 totally softened.

In 7 the grey substance of the corpora striata was of a violet or raw-flesh colour; in 2 that of the optic thalami was red.

In 2 the walls of the large ventricles were rugose by vascular expansions; in 1 they, as well as the central parts of the brain, were soft.

In 1 the pia mater of the cerebellum was infiltrated with pus; in 10 notably injected; in 5 it adhered to the surface of the hemispheres of the cerebellum.

In 4 the substance of the cerebellum appeared diminished in firmness; in 16 it was coloured lilac or violet.

The rhomboidal spaces were fibrous, and encrusted with lime salt in 1; in 10 the grey substance of the pons was a violet colour.

In 1 the spinal dura mater was covered with an extravasation of blood; in 3 the sinuses of the spinal cavity were much congested; in 1 the spinal prolongation was atrophied and disintegrated.

In 8 microscopical examinations were made. In two, the web of the cerebral pia mater contained many pus globules and pyoid elements; in another, it contained amorphous granular elements, or small

granular cells in course of formation. In the greatest number of cases the grey substance was not disintegrated; it was sometimes moist, and easy to spread out. It generally contained a more or less considerable ramification of vessels; almost always the external walls of the vessels were, as it were, sprinkled with greyish granules, or spotted with small finely granular cells. Sometimes the formation of these small cellules commenced in the very midst of the grey substance, where many small pointed spheres, like the eggs of insects, could be distinguished.

Delirium Tremens.—It is probable that the great differences of opinion that exist as to the proper treatment of this condition are due to the fact that we habitually use the term too generally, and as inclusive of morbid states only loosely connected together by the element of a common alcoholism.

M. Laval, however, in his study of delirium tremens, has arrived at the following conclusions:—

‘1. All delirium is due to a pathological process carried on in the intimate structure of the cerebral cell, starting with irritation, and leading to steatosis of the histological elements.

‘2. Alcoholic delirium, improperly called delirium tremens, only differs from delirium in general by its first cause, alcohol; seeing that the mechanism of its production, its symptomatology, its complications, its diagnosis, obey wholly the general laws which regulate other species of delirium.

‘3. Finally, digitalis, opium, chloroform, are no more specifics for delirium tremens than for other

delirious manifestations, and in all cases recourse must be had to a rational treatment. The patient, and not the disease, must be treated, the author regarding delirium tremens as not being a morbid entity.'

With the great diversity of views respecting this condition, it will be necessary to say a few preliminary words before speaking of the pathological anatomy.

Alcohol, when it morbidly affects the nervous centres, may do so in one or other of several ways.

1. It may do so with the phenomena of a fit of drunkenness, of which the symptoms differ in different individuals; in some, never amounting to excitement, the patient only becoming more and more stupid until he falls into a state of hebetude or even coma; in others, alcohol will induce the most frantic delirium, sometimes only for a few moments, at other times for many hours (*delirium e potu*). There are, of course, infinite shades of variety between these two states.

2. It may affect the patient periodically with the phenomena of a fit of dipsomania. In some people the tendency to drink to excess is persistent, in many others it is strictly periodic. I have a lady now under my care who has this tendency only during the catamenial period. The symptoms of this attack vary as much as those of ordinary intoxication.

3. The patient may manifest the symptoms of chronic alcoholism with indefinite cerebral phenomena, such as a tendency to emotional weakness, the being easily startled, a certain want of purpose in all the concerns of life, diminution of intellectual power, the impression of fear, certain morbid sensory phenomena,

excessive tremor at times almost simulating paralysis agitans, and often towards the close of life some motor paralysis. In some cases insanity supervenes.

4. Delirium may occur also in persons who, having been habitual drunkards, have been subject to some great shock, some accident or operation. This condition, called traumatic delirium tremens, may be anæmic delirium, or it may be reflex delirium, the alcohol only playing the indirect part of rendering the nerve centre more sensitive to excitation from a distant organ. At any rate it is certain that this traumatic delirium tremens may occur where there has been no indulgence in intoxicating liquors. Dr. Handfield Jones mentions several examples ; and he deduces from these examples the opinion that the peculiar morbid state (in delirium tremens) is not necessarily one of poisoning at all. The distinction between this nervous disorder and true delirium tremens is the more important that the two conditions often demand a diametrically opposite treatment.

5. We get true delirium tremens, manifested by various symptoms and various physical signs, but never manifesting itself by any intensification of pleasurable emotions. The emotions excited seem always to be either horror, or fear, or melancholy. Still I doubt whether the commission of suicide in delirium tremens is ever the result of melancholy. It is probably always an unreasoning attempt to avoid present horrors, such as the hallucinations of sight, constantly present to the view of the patient.

I am not going to give you all the symptoms of

delirium tremens. Suffice it to say, that the tremor varies immensely in different cases; that the patient often at the beginning of his illness seems preternaturally busy; that though the attempt to be busy passes away, the delirium all through the patient's illness may keep upon the ordinary avocations of his life; that the horrors may be completely absent, and all the hallucinations of sight and hearing be connected with the common business of life; that, on the contrary, in many, if not in most cases, the delirium will be connected with scenes of terror, the hallucinations with loathsome reptiles, the ideas (in Protestant countries particularly) with a present or a coming hell. One more point with reference to the mode of attack. It has formerly been taught that these phenomena occurred in habitual drunkards from a deprivation of the accustomed stimulus, and no less an authority than Dr. Handfield Jones, basing his opinion on observations and statistics of Dr. Marston, considers this former teaching to be the right one.

Dr. Marston showed that delirium tremens comes on in soldiers commonly from the first to the seventh day after the privation of liquor, and this in men who had not suffered from it before. The statistics of prisons, whose occupants are largely drawn from the class of habitual drunkards, go far to disprove this theory, as also does the observation of many cases in which the attack comes on during the full indulgence in alcohol. It is certain, however, that the phenomena either commence or are greatly intensified many hours, in some cases several days, after the deprivation; but

I think it is in accordance with observation to say that they are not caused by the deprivation. The effects of alcohol vary in different individuals. Perhaps no two persons absorb it with equal rapidity; still less do they agree in the time necessary for its oxidation. We shall see directly that it is not alcohol itself that causes the morbid phenomena so much as its chemical products; and when these are formed they will act upon the nerve-cells in different individuals with greater or less rapidity. It is perfectly easy to understand that in two people the alcohol will be broken up quickly in one and slowly in the other, and the products exercise a rapid action on one brain, a slower one on the other.

These few words seemed necessary before speaking of the pathological results in this disease. And here, as in insanity, and probably in other nervous disorders, the post-mortem appearances are not the only, if they are the main, test of the lesions. Here, too, as in insanity, the sphygmograph has proved of service, and the tracings show a close similarity to what is seen in fevers and inflammation of a typhoid type, and are especially remarkable for the prominence of the phenomenon called 'Dichrotism.' This is interesting in connection with Dr. Johnson's assertion of the frequent presence of fatty degeneration of the heart in these patients.

Dr. Fraser gives the following post-mortem appearances in fifteen cases of idiopathic delirium tremens:—

Head—

In 8. Ventricular and arachnoid serous effusion.

In 1. Sanguineous arachnoid effusion.

In 1. Subarachnoid gelatinous effusion.

In 2. The scalp and dura mater deeply injected.

In 2. The brain and membranes much injected,
and of a rosy hue throughout.

Lungs—

In 1. Emphysematous.

In 2. Gorged with pus.

In 7. Gorged with blood.

In 1. Tuberculous.

Heart—

In 3. Dilated.

In 1. Hypertrophied.

In 3. Fatty.

In 1. Effusion of blood in the pericardium.

In 1. Fibrinous clot in the pulmonary artery.

Liver—

In 7. Enlarged.

In 1. Fatty.

In 2. Cirrhotic.

Stomach—

In 5. Congested.

Kidneys—

In 1. Disorganised.

And he says there is no special appearance in either the brain, spinal marrow, or the membranes which can be placed as a constant effect of this disease ; for all the conditions which have been seen by the many observers have been noticed when there had been no delirium tremens, and all of them have been absent under the most aggravated and fatal cases of this disease.

I add the following cases, illustrating various post-mortem appearances :—

Case 1. Man, aged 33. Died rather suddenly after twenty-four hours of intense delirium. Treatment, digitalis.

No congestion of brain. All the encephalon rather pale. Brain wet. A good deal of subarachnoid fluid both on the convexity and at base. No fluid in the ventricles. Septum lucidum, pons, and cerebellum very soft. Arteries healthy. Both lungs very dark from congestion, but crepitant. Heart rather large. Several old white spots on right ventricle. Hardly any blood in the heart. Pulmonary and aortic valves with aorta and internal surface of left ventricle deeply stained. Walls firm. Valves healthy. Very little atheroma of the aorta. Liver large, hard, bloodless, and waxy. Gall-bladder full of bile. Spleen rather large. Kidneys healthy, except that the capsule of the right kidney did not peel readily. Internal surface of the stomach highly congested, and covered with ecchymoses.

Case 2. Man. Delirium tremens four days. Beer and opium treatment. Head: much sub-arachnoid fluid both of convexity and at base of the brain. Veins of hemispheres rather full of blood. The brain when taken out fell apart, most of the central white matter being very soft. The fornix, corpus callosum, and boundaries of the lateral ventricles were hardly of greater consistence than cream. No fluid found in the lateral ventricles, but it may have escaped when the brain was being taken out. Corpora striata and optic

thalami soft. Soft commissure, almost liquid. A little white thickening of the arachnoid at the base of the cerebellum. Pons and medulla oblongata healthy. Vessels of the base healthy. Thorax: heart with a good deal of fat externally; and the walls of the ventricles were partly encroached upon by, and partly converted into, fat. Walls of right ventricle very thin. A little atheroma round base of aortic valves. No clots anywhere. Fluid blood in both ventricles and in pulmonary artery. Lungs intensely engorged with blood; otherwise healthy. Abdomen: liver healthy, except that on convex surface there was a little cyst, the size of a filbert, full of bile. Kidneys healthy, except that the capsules split up, one layer adhering to the organ. Suprarenal capsules and spleen healthy.

Case 3. Man. Many attacks of delirium tremens; died in epileptic fit. Head: vessels of the dura mater and the brain much injected. Brain firm. Lateral ventricles enormously distended. The fluid they had contained had been let out in the process of getting out the brain. Kidneys healthy.

Case 4. Man, aged 32. Had delirium tremens for four days. Many epileptiform attacks, one every ten minutes during the last twenty-four hours of life. Head: dura mater healthy. Chronic thickening of the arachnoid over convex surface of both hemispheres, with opaque spots and plates. Much subarachnoid fluid. Brain somewhat atrophied; otherwise healthy. Kidneys and liver healthy.

Case 5. Man, aged 48. Tremor. Sleeplessness. Delirium. Almost fatuity. Head: all the vessels of

the brain engorged with black blood, both arteries and veins. No softening of brain. A little fluid in left lateral sinus. Much subarachnoid fluid. Arteries at base of brain rather atheromatous. Thorax: a spot of grey hepatization in lower lobe of right lung, about the size of the palm of a hand. It was perfectly circular. The rest of both lungs healthy. A little adhesion of both lungs to thorax. Heart covered with fat. Very little remains of muscular wall of right ventricle. Abdomen. Liver fatty. Kidneys rather granular; the capsules do not peel readily. Pelves of kidneys full of fat.

The presence of the fluid under the arachnoid, and perhaps even in the ventricles, is in consequence of the atrophic condition of the brain itself. This is one of the most frequent post-mortem appearances, the commencement of shrinking of the convolutions, due probably to insufficient nutrition by the alcoholised blood. Alcohol has been sometimes found free in the fluid in the lateral ventricles. The whole mass of the brain wastes. This takes place sometimes by way of fatty degeneration, at other times by gradual atrophy of the elements of nervous tissue.

The delirium cannot depend upon the arachnitis. The thickening of the arachnoid is generally so chronic, whilst the cerebral phenomena are so recent, that there cannot well be any connection between them. Besides which extreme chronic arachnitis may persist, without any cerebral phenomena whatever. The thickening of the arachnoid seems related to that gradual development of fibrous tissue that one sees in delirium tremens,

sometimes in the lungs, sometimes in the liver, or the kidneys, or the larger arteries, and occasionally in many organs together.

But post-mortem records point to more than the results in the brain. They not only show that these appearances are of an atrophic character, but they absolutely demonstrate the condition of blood that must lead to this atrophy. It is dark in all the vessels, and in some cases stains the endocardium as it does in some of the blood diseases. Now, Prout and Vierordt have proved that even a moderate use of alcoholic liquors causes both an absolute and a relative diminution in the amount of carbonic acid excreted. The increased excretion of carbonic acid which accompanies digestion is considerably checked by the use of spirits. If this is the effect of small doses of alcohol, what will be the effect of large doses taken daily, without any interruption or opportunity for more efficient decarbonization. The constant diminution in carbonic acid excreted gradually causes a sure augmentation of carbon in the blood; so that this fluid will sometimes contain as much as thirty per cent. more of this matter than in health, and will in some cases assume an oily or even a milky appearance. The renewed supplies of alcohol, creating an artificial stimulus to the motions of the heart and the frequency of the respirations, will add still more to the baneful carbonaceous accumulation. This fatty carbonaceous blood, unfit for the nutrition of any organ, is especially unfit for the due performance of those regulated physical conditions of osmosis in so fatty an organ as the brain. It must also lead to congestion of the internal viscera.

We see by this statement how far we can agree with Dr. Handfield Jones's statement, that delirium tremens, except in cases of acute occasional excess, is much more than alcoholic poisoning. Without binding ourselves too closely to humouralist views, it is only a fair result of observation to acknowledge that the direct and indirect effect on the blood of undue inhibition of alcohol is to load this fluid with a proportion of carbonic acid that not only alters its appearance, but materially modifies its nutritive powers and impedes the circulation. And this morbid blood may not only induce congestions of the internal viscera, or even low inflammation of some of them, such as the arachnoid, the lungs, the liver, the spleen, or the kidneys, but by its innutritious character impresses that atrophic modification on the brain, that is shown *post mortem*, leading to the compensating increase of the subarachnoid fluid.

In addition to these lesions the arteries of the base of the brain are sometimes found atheromatous, and with such alteration in the lumen of the vessels that thrombosis is easy, and softening as a necessary consequence. Nor are other changes altogether wanting. Small granular kidney is not a very unusual concomitant of delirium tremens, and the vessels of the brain may therefore have to bear the additional pressure of the lesions consequent on deficient excretion of the urinary constituents.

Dr. Fraser makes another suggestion as to the mode of blood-poisoning in this disease. He says : ' Alcohol is said to take the oxygen of the blood (which would otherwise have gone to the normal oxygenation of the

various tissues of the body), and thus to stop the ordinary waste of material, while its carbon and hydrogen are given off as carbonic acid and water. Why, then, if the action of alcohol merely stops waste, should its action be so injurious? Because the arrest of this waste throws into the circulation an abnormal amount of nitrogenous matter. The resemblance between the convulsions attending delirium tremens and those attending uræmic poisoning is close, and arises either from a similarity of action of the alcoholic and uræmic poison on the nervous centres, or from a retention of urea during the delirium tremens, such as happens in true uræmia.

Observations are wanting on the spinal cord in this disease. It is impossible, with this atrophic modification of the brain, but that something similar should obtain in the spinal cord. That such lesion is of no great severity is rendered certain by the absence of all paralysis in most cases, and by the subsidence of tremor after prolonged sleep. But the explanation of the tremor is yet wanting. It cannot depend on sclerosis of any portion of the encephalon, or it would not disappear so rapidly. It must probably be from the malign influence of, or the negative fact of, mal-nutrition by the carbonaceous blood of some of the motor centres, either some of the external cerebral convolutions, or some large organs at the lower part of the brain, or the great cells of the grey matter of the spinal cord.

Whatever may be the true explanation of this motor phenomenon, it is to alcohol and its effects on the brain

itself, with the secondary results of the circulation of this innutritious fluid through the brain, that we must look for the pathological lesions of delirium tremens, and the evils of this impure blood are in many cases intensified by the concomitant lesion of fatty degeneration of the heart.

Consult—

Fœderé, 'Traité du Délire.'

Calmeil, vol. i. 237, op. cit.

Graves, 'Clin. Lectures.'

Handfield Jones, 'Functional Nervous Disorders.'

Reynolds, 'System of Med.'

'Med. Chir. Trans.,' vol. xlviii. Dr. Weber.

Wilks, 'Path. Anatomy.'

Dr. Peddie on 'Pathology of Delirium Tremens.'

'Lond. Hosp. Rep.,' vol. iii. Dr. Fraser.

'Med. Record,' i. 296. M. Laval.

LECTURE VII.

INSANITY.

It has been said by no less distinguished an alienist physician than Griesinger that 'if we examine the great mass of uncertain records, and except the cases in which the insanity was cured before death, there still remains a number of cases, reported by careful observers (and which may be confirmed in any asylum), where the cranial cavity and its entire contents presented altogether normal relations.'

In a vast number of cases of insanity there may be found recognisable lesions. The more precise our methods of investigation become, the better are we able to appreciate the finer structural changes in the nervous centres. But even when the microscope has done all that we can expect of it, there still remain, and there always will remain, instances in which, while the symptomatic phenomena are most intense and startling, yet it is impossible after death to discover structural alteration. Are these cases instances of functional disease? Well, that leads me to ask a question peculiarly important in the consideration of nervous diseases; is it possible that any symptom can occur without organic change; or is so-called func-

tional disease a condition that merely leaves no apparent alteration of structure *post mortem*. Even in these nervous diseases, and they are the most difficult of all, you will be right in assuming that morbid phenomena are invariably associated with organic change, but that in a number of cases this organic change is not of a nature to persist after death.

There are, at least, four conditions that may induce very serious cerebral symptoms, and yet leave little or no change of structure to be recognised after death: 1, a condition of the blood itself, as in uræmia, spanæmia, phthisis, &c.; 2, a variation in the normal blood supply to the brain; 3, reflex irritation, though this perhaps might be classed under the second head, as irritation radiating from some distant organ would be likely to induce, by reflex action, contraction of the arteries; 4, shock. Each of these, if long continued or frequently repeated, will induce structural lesion that can be recognised after death; each of them may be the starting point of phenomena of a severe character, and if the duration of the attack be not protracted, will leave no post-mortem appearance. Still the lesion is present during life. It may be a condition of blood wholly unfavourable to osmosis; it may be a transitory congestion, or an anæmia, partial or complete, from contraction of the arteries; it may be a total paresis of absorption.

Dr. Thompson, of the Bristol Borough Lunatic Asylum, has done some good work here by means of the sphygmograph. He finds by observation on the pulse tracings of general paralysis that this condition is

marked in very early stages by persistent spasm of the vessels ; that this persistent spasm will lead to changes in the component elements of the vessels, especially in the muscular substance ; and further, that the sphygmograph, by indicating the true nature of the disease at a period when it could barely be suspected by other symptoms, affords an opportunity of applying remedies—the calabar bean, for instance,—when mere function is disturbed, before actual change has begun, and when the remedial means can be of the most avail.

This is an illustration of a morbid condition that at first would leave no recognisable structural results ; but it would be wrong to say that the early symptoms of general paralysis were not connected with organic change. The organic change is in the diminished calibre of the vessels, which not only persists, if left untreated, but will lead to further lesions ; whilst, if recognised early, it is amenable to remedial means, at least for a time.

It is certain that the same may be said of the early stages of mania and melancholia. The true pathological anatomy of these first beginnings of mental disease is to be sought for during life, and most of the structural lesions found *post mortem* are secondary results ; secondary, that is, not to the morbid mental phenomena (such a statement, though it has been made, is eminently ridiculous), but secondary to the early organic changes that are only of temporary duration.

It having, then, been insisted on that the early phenomena of several varieties of insanity depend upon lesions always present, yet transitory in their duration,

often amenable to treatment, and from their very nature not recognisable after death, it remains to study the coarser structural changes induced by their earlier lesions.

The majority of post-mortem examinations of the insane show anatomical changes of some kind or other ; and, as Griesinger says, there are certain structural diseases of the brain which always cause considerable anomalies in the mental functions, even insanity. A diffuse inflammation of the grey substance, extending over a number of convolutions, has never been observed without profound mental disturbance ; extreme meningitis of the convex surface (in previously robust individuals), considerable acute œdema of the greater hemispheres, rapid bilateral atrophy of the convolutions, a deeply penetrating alteration of the ventricular surface of any extent, were never observed without psychical disturbance, particularly mental weakness.

The examination of a brain should always be made, first, in the fresh state, and afterwards from hardened sections.

It may be convenient, first, to touch upon the alterations found in the various elements of the nervous tissue, the vessels, the cells, the neuroglia, mentioning the morbid conditions of the cranium and the membranes ; and secondly, to pass in brief review the most common lesions found in mania, melancholia, dementia, general paralysis, and idiocy.

Although we may somewhat cavil at the universality of Rindfleisch's dictum, that we must concentrate our attention almost entirely on a chronic hyperæmia of

the cortical substance as the common foundation of all further mischief, yet a very considerable amount of recognisable lesion in insanity is connected with the vessels. Abnormal dilatation and abnormal contraction may exist during life, and leave no mark behind. More commonly the vessels of the membranes, or of the brain, and often of both, show very definite lesion. The larger vessels of the membranes and the vessels at the base of the brain are often atheromatous and calcareous; but it must be remembered that many of the autopsies in asylums are held on aged persons, in whom this kind of degeneration is common, and that this condition has more to do with age than with insanity. Atheroma is seldom found in the vessels of the cortex at the upper portion of the brain. The vessels of the pia mater are not only very frequently congested, but have been the seat of inflammation; and a low form of meningitis, involving the cortical layer of the brain, is not unusually met with.

But whether this hyperæmia has gone on to inflammation or not, it will, if frequently repeated, have left its marks both on the vessels and on the brain.

The changes that occur have been recorded in a masterly sketch by Dr. Batty Tuke, from whose observations I gather much for the present Lecture. He calls attention to a fact already recognised, that this hyperæmia, leading to dilatation of vessel, causes expansion of its surroundings. When the vessel is relieved from pressure it contracts to its original calibre, and leaves a space. This had been already alluded to under 'hyperæmia' in Lectures II. and

III.; and it was there stated that little pits or holes, *l'état criblé*, were often found, caused by condensation of brain tissue, due to the pressure of the dilated vessel and subsequent exudation. This space may become the receptacle of lymph thrown out in subsequent congestions; and evidences of this exudation are found in a thickened condition of the hyaline membrane, an evident induration of the contiguous brain, and considerable deposit on the adventitia.

The thickened condition of the hyaline membrane, the hyaline-fibrosis of Drs. Gull and Sutton, is a lesion of common observation, and wholly irrespective of chemical re-agents in the preparation of the microscopical specimen. It is, however, not by any means specially connected with venal disease; it is a condition often found in old age, and seems to depend on previous dilatation of the vessel and exudation from it.

Deposits on the tunica adventitia are also found after prolonged hyperæmia. Dr. Sankey first described a peculiar molecular material, not unlike oil in appearance, but not answering to the chemical tests for fat. Dr. Batty Tuke has found this material in every brain of sane or insane persons that he has examined, but much more largely in the insane, and especially in old cases. It is probably the result of transudation. Besides this material, crystalline masses of hæmatoidine are found, especially in the angles of bifurcation of the vessels, in all forms of insanity. Minute extravasations of blood are frequently found, and depend upon a variety of causes. In some cases minute aneurisms

have been seen, and Rindfleisch states that these punctiform hæmorrhages generally are due to aneurisms of the smallest veins. But extravasations occur from the giving way of the distended vessels in various states of disease, fatty degeneration, atheroma, &c. I believe aneurism of the pia mater to be more common than of the vessels in the interior of the brain; but Dr. Tuke has found no less than five aneurisms in the right corpus striatum, immediately underlying the epithelium in one case.

We also meet with a change of the extravasated blood or of the hæmatine into pigment, the colouring matter being deposited outside the vessels.

Fatty degeneration of the capillaries is an occasional but not a constant lesion in insanity.

Hypertrophy of the muscular coat is met with pretty frequently, and it has been variously interpreted. Dr. Sankey believes it is due to increased exercise of the vessels in consequence of an impure state of the blood. Dr. Tuke suggests that the degeneration of the cells of the sympathetic ganglia, so constantly noted in cases of chronic insanity, is more likely to be indicative of this change. It is not really the result of an attempt on the part of the distended vessels to contract on their contents.

It is interesting to note that hypertrophy of the muscular coat is always accompanied by the hyaline-fibrosis just spoken of, although this latter condition often exists independently of any hypertrophy of the muscular coat.

And lastly we have some alterations in the direction

of the vessels ; undue straightness due to the distension ; tortuosity or kinking caused by the withdrawal of the strain.

Taking next the membranes, we find in the dura mater evidences of old inflammatory processes, thickening of the dura mater, and partial or complete adhesion of it to the bones of the skull. In the arachnoid we meet with the same difficulty in insane persons as in other morbid conditions of this membrane, viz., that it is so intimately connected with the dura mater and the pia mater. It is frequently opaque and thickened in all forms of insanity, or adherent to the dura mater, or to the pia mater, or with bony plates on its surface, or with fine granulations on the external surface, or on the ependyma of the ventricles, evidences of chronic inflammatory action. In addition to these lesions, small ecchymoses are found in the cavity of the arachnoid, and Virchow says pseudo-membranes, though these have been explained by other observers as extravasations of blood with absorption of the hæmatine. I have no doubt the former explanation of Virchow is the true one. The most peculiar affection of the arachnoid in the insane is seen in the hæmorrhages into its sac and their results. These may be seen in their recent condition, and will then be simply masses of blood, of greater or less extent, compressing the brain, and causing abnormal separation between the layers of the arachnoid, though generally the hæmorrhage is slow and gradual. Very soon, however, these masses of blood undergo change. Some of the fluid part is absorbed, and the lymph forms a cyst-like wall adherent

to the layers of the arachnoid, being firmly united to them, and becoming organised, enveloping either a clot or serum, or small accumulations of blood globules, and forming what are known as arachnoid cysts. Dr. Sutherland says the cysts may take the form of a small sheath, not unlike the finger of a glove in size and shape, or may consist of two large flat walls of organised lymph, which may be thick or thin, and divisible or not into laminæ. The cysts may be so soft that they cannot be removed without tearing, or of such strength and toughness that they will bear rough handling, or can be filled with water and suspended by a cord without their walls giving way. They are generally thick in the middle; but become more attenuated towards the edges, where the two walls adhere to one another, forming an acute angle. They are found on both hemispheres, and generally, but not always, on the upper surface. They are often due to blows on the head, but may form, without previous accident, from hyperæmia of the meningeal vessels, with weakness of the vessels themselves, and probably impoverishment of the blood. Idiocy, dementia, and general paralysis are the forms of disease most usually associated with these cysts of the arachnoid; but, like many other cerebral lesions, they are found in persons who have not been insane.

It is a moot point whether the external layer forming the cyst wall is the peripheral layer of the coagulated fibrine, or the fibrinous exudation of an inflammation originating secondarily around the clot. Most commonly I believe the cyst wall is formed by the coagulated fibrine itself.

Dr. Bright gives a beautiful plate of one such cyst which contained half a pint of serous fluid.

In the pia mater we may find active hyperæmia of the vessels, and dilatation of the vessels in mania compared with the normal size; hæmorrhages of various amounts, frequently punctiform, due, as in the case of the cerebral vessels, to small aneurisms, to atheroma, to fatty degenerations, &c., all the various changes in the structure of the vessels that have been mentioned as occurring in the vessels of the brain; a special congestion connected with the larger veins, leading to thickening and œdema of the pia mater; sometimes a very anæmic state of the pia mater as part of a general anæmia; and a thickening of the whole membrane from inflammation, which generally involves some of the outer layers of the cortex of the brain, so that in attempting to strip off the pia mater the external layer at least of the brain's substance comes with it. This condition as it affects the brain will be spoken of immediately. Deposits of crystals of phosphate of lime have been found by Dr. Tuke in one case, the subject of acute melancholia due to great brain exhaustion; and lastly the same observer has met with two cases, in which there were lymph deposits between the pia mater and the substance of the spinal cord.

The ventricles are often found dilated, doubtless a secondary lesion consecutive to atrophy of the brain. Of very little importance also are the alterations in the shape of the ventricles from partial contractions and shortening and adhesions of their surfaces. Bergmann observed adhesion of the posterior cornua in several

hundred cases, and considered this condition to be the special pathological lesion in chronic dementia, but it is not unusually met with in the brains of sane persons.

Vascularity, and even inflammation of the pineal and pituitary glands, have been met with, and we shall see by-and-by that a morbid state of the latter organ has been found in many cases of epilepsy; but observations on these glands seem to have no definite relation with the phenomena of mental alienation.

I have already mentioned many of the changes the neuroglia undergoes in the insane while speaking of the various forms of sclerosis, Lecture IV.

General sclerosis may be met with congenitally, or in some cases of idiocy, or in a mental condition that can only be described as one of hebetude. It is not common.

Disseminated sclerosis is very constantly found in general paralysis, and in many cases of chronic insanity of various forms. It may occur as true sclerosis, or as grey degeneration with corpora amylacea.

Miliary sclerosis is also common in general paralysis. The neuroglia, too, is much atrophied in cases of atrophy of the nervous centres depending on impaired nutrition, due to atheroma of the vessels. In this morbid state all the elements of the tissue are seen to be diseased, cells, nerve-tubes, and neuroglia.

Another form of disease of the neuroglia is colloid degeneration.

It is not associated with proliferation of the nuclei, but is a form of degeneration of the nuclei of the neuroglia.

It is found in cases of chronic insanity, and is most marked in the white matter. Griesinger speaks of the colloid masses and corpora amylacea being one and the same thing. The difference of their behaviour with carmine shows some distinction between them. The parts affected in each case are the nuclei of the neuroglia, but the form of degeneration is different in the two cases.

The condition of the cells may vary according to the special disease. It is at present difficult to determine, whether any abnormality of cells can be the primary lesion in mental disease. Most probably all alteration in their number and condition is secondary to the vascular abnormalities previously referred to. Still, as secondary lesions, the alterations in the cells are very noticeable. These lesions consist of atrophy of cells, especially in the external layer of the cortex, diminution in their number, or even in spots, entire absence. Drs. Tuke and Rutherford, and after them Dr. Herbert Major, have described considerable enlargement of the cells of the inner layers of the cortex, where the cells of the outer layer were affected by degeneration. In senile insanity, old dementia, and especially in general paralysis, the cells are found pigmental and granular. In other cases there is complete degeneration of the cell-wall, with a setting-free of the nucleus.

Speaking more generally of lesions in the insane, we sometimes find an apparent hypertrophy of brain; it being impossible, when the skull-cap is removed, to get the brain back again into its normal cavity. It is

very rare, and, as was said in Lecture IV., it is not due to general hypertrophy of all the elements of brain tissue. Still, when it is associated with a dry condition of membranes, anæmia of the whole brain, and some flattening of the convolutions, with empty ventricles, the increase in volume must be due to abnormal increase of some portion or other.

The cases are so rare, that it is mere hypothesis to suggest that in all such instances this increase is due to great proliferation of the neuroglia nuclei, but this is the lesion that has been found in the hypertrophy of brain in children.

Atrophy is extremely common in chronic cases, and it is impossible by the naked eye alone to distinguish between senile atrophy and the form that follows very acute disease. In both the membranes may be thickened; in both there is great œdema of the pia mater, varying in exact proportion to the atrophy of the convolutions; in both the sulci are wide and distended with this fluid, and the brain as a whole smaller than natural.

The shrinking of the convolutions may be universal, or more in one part than in another, of one hemisphere, or of one convolution. Partial atrophy, however, is far more common as the result of previous disease (chronic mania, dementia, &c.) than of old age.

I refer to the remarks on atrophy, Lecture IV., for an explanation of the causes of this lesion; but partial atrophy is frequently associated with sclerosis, or is due to the absorption of spots of hæmorrhage or softening.

Without giving you all the conflicting observations as to the weight of the brain in the insane, it may suffice to point out these results :—

1. The weight of the whole brain (cerebrum, cerebellum, pons, and medulla oblongata) is somewhat less in the insane than in the sane, regard being paid to the age of each. This is particularly seen in chronic cases.

2. The weight of the cerebellum, pons, and medulla oblongata is rather more in insane males than in the sane.

3. Therefore the loss of weight of the brain in insane males is at the expense of the cerebrum proper.

4. The weight of the brain does not always bear any exact proportion to the intellectual power.

5. In brains, whose weight is not above the average, the depth of the grey matter may account for the intellectual power.

6. With the exception of idiocy, no particular mental disease is associated with a small or a light brain. Thus in Dr. Clapham's cases the maximum male brain was found in connection with senile dementia, and the minimum with dementia; the maximum female brain occurred in acute forms, the minimum in epileptic insanity.

7. Dr. Bucknill finds an average lower specific gravity in the brains of the insane. He even thinks there is a kind of atrophy of the brain in mental disease, which manifests itself solely by diminution of the specific gravity, a kind of fatty degeneration of the cerebral substance without diminution in volume.

Mr. W. G. Balfour has collected from the records of the Montrose and Fife and Kinross Asylums, and from observations of his own at Colney Hatch, the post-mortem appearances (not microscopical) in 700 insane cases. I cannot give you a better idea of the most usual lesions than by quoting these results:—

Number of cases, 700.

Calvarium abnormally thickened . . .	in 112
„ „ thin . . .	59
„ dense . . .	12
„ loose in texture . . .	9
Caries and perforation . . .	1
Membranes. Dura Mater:	
Adherent to calvarium . . .	74
Abnormally thickened . . .	76
Calcareous deposit . . .	1
Abnormally thin . . .	1
Ossification of . . .	7
Tumours attached to . . .	10
Arachnoid:	
Effusion of blood into sac of . . .	27
„ „ serum . . .	125
„ „ pus . . .	2
False membranes in sac of . . .	38
Sanguineous cysts . . .	3
Crystalline granulations . . .	19
Opacity of . . .	294
Adhesion of surfaces of . . .	6
Pia Mater:	
Marked injection of vessels . . .	53
Local bullæ of fluid in meshes . . .	61

Sanguineous effusion into meshes . . .	in	41
Tumours attached to	„	3
Brown gelatinous deposit	„	24
Adhesion to surface of grey matter . . .	„	63

Blood vessels of brain :

Atheroma of arteries	„	108
Aneurismal dilatations	„	3
Marked injection of	„	59

Grey substance :

Abnormalities in thickness of	„	35
„ „ colour	„	79
Gelatinous softening of	„	10
Effusion of blood into	„	6
Granulations of surface of convolutions . . .	„	3
Acute inflammation of }	„	10
Local atrophies of }		

White substance :

Induration of	„	39
Œdema	„	75
Softening	„	31
Effusion of blood into	„	15
Excessive shrinking of	„	30
Marked injection of	„	64
Cysts in	„	6

Optic Thalami and Corpora Striata :

Sanguineous effusion into	„	19
Softening of	„	25
Tumours in	„	3
Cysts in	„	7
Cicatrices in	„	6

Cerebellum :

Adhesion of membranes to	.	.	in	3
Effusion of blood into pia mater of	.	„		5
„ „ serum „ „	.	.	„	1
Hæmorrhage into substance of	.	.	„	2
Softening of	.	.	„	1
Cysts in	.	.	„	1

Ventricles :

Excessive serous fluid in	.	.	„	353
Sanguineous effusion into	.	.	„	20
Opacity of	.	.	„	68
Softening of floor of	.	.	„	4
Cysts in choroid plexus	.	.	„	70
Tumours in choroid plexus	.	.	„	2
Earthy deposit in choroid plexus	.	„		12

Pons : granulations on free surface of . „ 1

Corpora Quadragemina : Softening at fissure of Rolando on left side . „ 1

Marked difference in size of the two hemispheres . . . „ 8

Apparently normal brains . . . „ 60

It is to be remarked in this, as in all similar collections of cases, that tumours are seldom found in the brains of the insane. Here they have been found three times in connection with the pia mater and twice in the choroid plexus, ten times connected with the dura mater, and only three times in the optic thalami and corpora striata; whilst apart from these organs the grey and white substances of the brain have wholly escaped. The proportion of cases is no larger

than would probably be found in an equal number of sane persons.

Dr. Boyd states that, during his residence in the St. Marylebone Infirmary, in three years and a half there were in 1,039 post-mortem examinations twenty-two cases of tumours in the brain; one of these was transferred to the insane ward, and two were in a state of fatuity, leaving nineteen, or at the rate of about 18·3 per 1,000, in whom no mental derangement was observed. Whilst he was at the Somerset County Lunatic Asylum, in 875 post-mortem examinations there were fourteen cases of tumours of the brain and membranes, or at the rate of 16 per 1,000.

Whilst it would not be scientific to speak of syphilitic insanity as a variety of madness, yet it is certain that morbid mental phenomena coincide with, if they do not depend on, syphilis. Thus headache, sleeplessness, with the symptoms of progressive dementia, with epileptiform or apoplectiform attacks, sometimes preceded by a period of maniacal excitement, sometimes devoid from the first of all excitement, but taking the form of melancholia and hypochondriasis, and especially of syphilophobia, and accompanied in its later course with paralysis of one or more of the cerebral nerves, and even with general diminution of the motor power, are phenomena that are very usually associated with syphilis.

Nor do these symptoms necessarily occur at a long date from the primary infection. They are known to have occurred within a month from the primary infection. It is on this account that Dr. Wille has con-

sidered these morbid symptoms may in some cases depend on anæmia rather than on any structural lesion. He would distinguish the following forms of mental disorder due to syphilis :—

1. Irritative forms based upon cerebral anæmia following syphilitic infection even from its commencement.

2. Simple inflammatory forms due to meningitis and inflammatory softening of the cerebral substance.

3. Neoplastic forms proceeding from cerebral or meningeal gummata.

I doubt whether any form of meningitis can by its appearance alone be accurately deemed specific, without the presence of gummata. Griesinger declares a partly diffuse, partly circumscribed thickening of the arachnoid to be specific; while Maudsley and others regard as specific the diffuse exudation glueing the membranes to each other, to the cerebral substance on the one hand, and to the skull on the other. It is probable, however, that the meningitis of syphilis cannot be distinguished from that accompanying rheumatism or other morbid states. It is evident, when meningitis is accompanied by paralysis of any of the cerebral nerves, that it is of the base of the brain; and it may reveal its presence by the effects on the optic disc. See Lecture XIV.

Syphilitic meningitis is sometimes secondary to caries of the cranial bones. In this case it is generally very partial.

Much that belongs to syphilis affecting the nervous centres has been said in Lecture V. upon tumours;

but we should notice a peculiar form of syphilitic encephalitis congenital and associated with mental phenomena. It probably begins as an inflammation of the neuroglia, either solely or coincidently with the connective tissue of the capillary vessels; and if life is prolonged, it may lead to general sclerosis of the whole brain, or of one hemisphere, &c.

Many of the morbid cerebral symptoms seem to be connected with the presence of gummata, or of small local softenings dependent on syphilitic thrombosis; but gummata may exist in the brain without giving rise to any mental symptom, or indeed to any phenomena except pain, and I have elsewhere in these lectures recorded such an instance.

It remains only to say a word about the cranium.

Almost every malformation of the cranium may be associated with idiocy, but microcephalous heads are most commonly found with this disease. In cases of chronic insanity the skull-cap is often thickened and indurated. Sometimes the bones are very thin, especially where they have been pressed against by a gradual accumulation of fluid in the ventricles. They may be carious from struma or syphilis, or they may be the seat of nodes externally or internally, of exostoses, spiculæ, &c.; and lastly, many injuries to the cranial bones have been known to have been the starting-point of morbid mental symptoms. Various abnormalities of the spinal cord are found in chronic insanity; myelitis, grey degeneration, disseminated and miliary sclerosis, &c. Such lesions are accompanied by ataxic or paralytic symptoms.

LECTURE VIII.

INSANITY (*continued*).

MANIA. It is not easy to draw pathologically accurate distinctive lines of demarcation between the various forms of insanity. Between mania and melancholia, especially, the pathological difference may be very slight. It is only possible, therefore, to give the most common appearances, whilst we acknowledge that much of the lesion we point out as belonging to mania obtains also in melancholia. The clinical history of each form of disease shows also that in many cases the lesions on which the symptoms depend are transitory, and not persistent. Not only do many patients recover both from melancholia and from mania, but in recurrent mania the attacks are separated by intervals, of variable duration, of perfect health; and in some cases the patients themselves can foresee the recurrence of the mental phenomena by their own cerebral sensations. Clinical observation, therefore, as well as pathological research, leads us to consider lesion of the vessels as at once the primary and the most important of all the cerebral changes in mania.

The post-mortem manifestations of cerebral hyperæmia are often not readily recognisable. The empti-

ness of the capillaries, as we have already said, is no proof they have not been distended during life, as the cerebral vessels are able to empty themselves with such facility. It is this capillary distension, this hyperæmia of the cortical substance of the brain, that is the chief lesion in acute mania. This hyperæmia will generally affect the pia mater, and, I believe, specially the pia mater of the convexity. Rindfleisch says that this cortical hyperæmia will here cause a sort of stasis; this, again, leads to over-distension, then to atony of the vessels. A return to the normal calibre of the vessels is at first only a question of time; it is only when the distension has become habitual that corresponding changes are set up in the walls of the vessels, and the return to the normal calibre becomes impossible.

I must risk the danger of being charged with repetition if I again remind you that this hyperæmia of the pia mater and the cortex may be shown merely in a slight tinge of redness; more frequently, however, its previous presence is manifested by its results. These are extravasations, diffuse encephalitis, affecting specially one layer of the cortex, and pigmentation; and if the hyperæmia has been long continued or has frequently recurred, further changes are found to have taken place in the vessels themselves. The extravasations may take the form of punctiform hæmorrhages, but more usually the extravasation has not absolutely reached the brain matter, but exists in the form of dissecting aneurisms of the small veins. Besides these minute aneurisms we find various dilatations of the smallest vessels, causing altera-

tions of shape of variable intensity. Dr. Bucknill thinks that in acute mania extravasations of blood are chiefly in the pia mater. Greding states that the choroid plexus was healthy in only 16 out of 216 cases of insanity, and that out of 100 maniacs 96 showed a choroid plexus that was either thickened or full of hydatids; by hydatids he doubtless means serous cysts. The inflammatory condition met with in mania is usually confined to the middle layer of the cortex; the external layer is occasionally affected, this layer of the cortex coming off in patches when the pia mater is removed, and leaving the appearance of ragged ulcerations of the external portion of the brain. The internal layer is scarcely ever affected in mania. These three layers correspond to the arrangement of the vascular branches in the cortex. The middle layer is in a state of red softening, and when it is thus affected the removal of the pia mater will often bring away the external layer pretty much as a whole, its cohesion with the softened middle layer being destroyed. I speak of three layers with a knowledge that Lockhart Clarke has divided the cortex into eight layers. For practical purposes, and especially as a basis for pathological observation, the smaller number will suffice, even though each one of these three layers may be further minutely subdivided. It must, however, be remembered that this important lesion, the softening of the middle layer, is not universally met with in acute mania. Were it so, we might be tempted to look at lesion of this middle layer as causing an ataxy of the brain corresponding to locomotor ataxy

of the muscles, the thoughts being there, but the power of combination being absent. We often get in acute mania an overwhelming richness and flow of ideas; the brain may react on the external stimulus with a marvellous quickness and ability, or it may be so taken up with its fulness of ideas that external stimuli pass wholly unnoticed. What is wanting is sequence, or at least such sequence of ideas as can be recognised. Much of the so-called incoherence of acute mania is no more than a rapid jumping from a first idea to a second and third, the first and third being put into words, the second, the connecting link, being passed over in silence. If a perfectly sane man, with his brain in full activity, were to put into words each idea as it arose, or still more, if he were to put into words every alternate idea, his speech would frequently simulate pretty closely the incoherence of acute mania. Inordinate richness of idea, without a sequence that can be appreciated by another person, may be considered one of the most important symptoms of acute mania; and this symptom may be connected with the hyperæmia of this middle layer of the cortex before further degenerative change has passed upon it.

But although this may be the case roughly, pathological histology will demand greater accuracy. It is not enough to localise the lesion in the middle layer, even if this were always the part affected. This middle layer itself contains no less than three varieties of corpuscles, viz., neuroglia corpuscles, small finely granular bodies having nuclei, and the pyramidal nerve-cells. In disease, not only does the proportion of these bodies

vary, but their outline and appearance; and it is on their normal proportion and freedom from all lesion that clearness of thought and systematic sequence of idea probably depend.

Pigmentation is a proof of chronicity to a greater or less extent. The blood corpuscles or the hæmatine extravasated either beyond the vessels or between the coats of the vessels is changed into pigment and connective tissue, and the part so affected is somewhat firmer than in health, but also stained a brownish colour.

And finally, in acute mania, where the symptoms have recurred often or have extended over some time, the distension of the vessels leads to still further changes, and there occurs fatty degeneration of the capillaries. It is not met with universally in these cases, but, when there, it imports an element of chronicity, or more or less of hopelessness of cure, on the case. It is doubtful whether capillaries can ever become normal in which oil globules collect to a considerable extent round the nuclei, such oil globules not being derived from the fatty portion of the brain itself, but being a sign of degeneration due to the long-continued distension of the capillary walls.

Melancholia. — The post-mortem appearances of acute melancholia are in many cases the same as in acute mania, viz.: dilatation of vessels, extravasation, subacute inflammation, and pigmentation. Extravasations occur more frequently in the pia mater than in the cortex. In a large number of cases the lesion is simply dilatation of the vessels. We judge so by the constant return to health of melancholiacs.

The symptoms of unreasoning depression, however, may coincide with very various lesions of the brain; with tumours, with syphilis, with the circulation of impure blood in the brain, with anæmia as a sequence of epilepsy, and even with the gradual formation of cerebral abscess.

Mania and melancholia may merge into each other. Many cases commence with symptoms of melancholia, at first gentle and quiet, then suicidal, which will afterwards advance to acute mania, with homicidal phenomena. In such cases it would be impossible to draw any pathological distinction between diseases that differ only in name.

In chronic melancholia, however, we often meet with further disease of the vessels; either the fatty degeneration of chronic mania or ossification of arteries. An anæmic condition of the brain is very common; and Griesinger mentions a consistence of the cerebral substance abnormally great, with more or less serous infiltration.

In very chronic cases, however, we may meet with the lesions of brain-wasting, large sulci, much cerebro-spinal fluid over the cortex, with the alterations both of vessels and of cells that will be mentioned below.

Chronic Insanity, Dementia.—We find thickening and opacity of the membranes the result of chronic stasis and slow inflammatory action. The dura mater may be adherent to the skull; and there is often serous infiltration of the pia mater and dropsy of the ventricles. There is sometimes superficial induration and adhesion of the pia mater: but this lesion is not

found in senile atrophy. We meet also with anæmia, loss of colour, and atrophy of the convolutions, a widening of the sulci between the convolutions, pigmentation of the external layer of the cortex, with sometimes induration of the cortical or of the medullary substance.

The minute changes causing some of the coarser ones are, according to Rindfleisch, an increase in the amount of the perivascular protoplasms, appearance of a number of fine feebly-refracting processes of protoplasms, giving the vessel a curious thorny appearance. Some atrophy of the ganglion cells is found; brownish discolouration of them, due to the imbibition of hæmatine. Sometimes fatty degeneration of these cells. Fission of the neuroglia nuclei is found in all the layers of the cortex. Connected with this, and leading to the induration above mentioned, forming the most important lesion in chronic mania, is the interstitial growth of the connective tissue of the cortex.

Frequently also we meet with a granular condition of the ependyma of the ventricles; and occasionally some of the spinal lesions above mentioned, hæmorrhage, myelitis, sclerosis, &c.

Dr. Crichton Browne says of senile dementia, that there is thickening of the vessels of the cortex by fibroid degeneration, and atheroma of the arteries at the base in some cases; whilst cerebral wasting is the true anatomical basis of the fatuity of old age. The coarse changes in the brain discernible with the naked eye correspond with the more intimate alterations in its structure. The microscope shows degeneration and softening. The cells of the grey matter are compara-

tively few in number and small in size, as if shrunken ; or, on the other hand, they contain molecular fat and pigment in an advanced stage of the disease ; aggregation of granules remain as the débris of distinct cells. The vessels traversing the grey matter are studded with fatty granules and large compound corpuscles, and the neuroglia pervading it also shares in the fatty change. The fibres of the medullary substance also undergo some degree of fatty metamorphosis.

General Paralysis.—The commencement of this disease, like some other forms of insanity, is connected with no lesion that can be recognised after death. Dr. Thompson's valuable observations with the sphygmograph have been already referred to in Lecture VII. He proves that general paralysis of the insane may be presumed to be owing, to a considerable extent, to persistent spasm of the vessels, and that this spasm leads to change in the component elements of the vessels, especially in the muscular substance. It is only the secondary effects, therefore, that we see *post mortem*.

But what causes the spasm? Observations such as these of Dr. Thompson are most important as carrying us one step further back towards the origin of the disease ; but this origin is not yet reached. It is all hypothesis that blood in a particular state has some effect on the vaso-motor nerves that we call 'irritation,' and that this irritation causes spasm. We do not know for certain that the blood has anything to do with it. We have not the slightest idea of what irritation is. It is a convenient word by which to formulate our ignorance, and that is all.

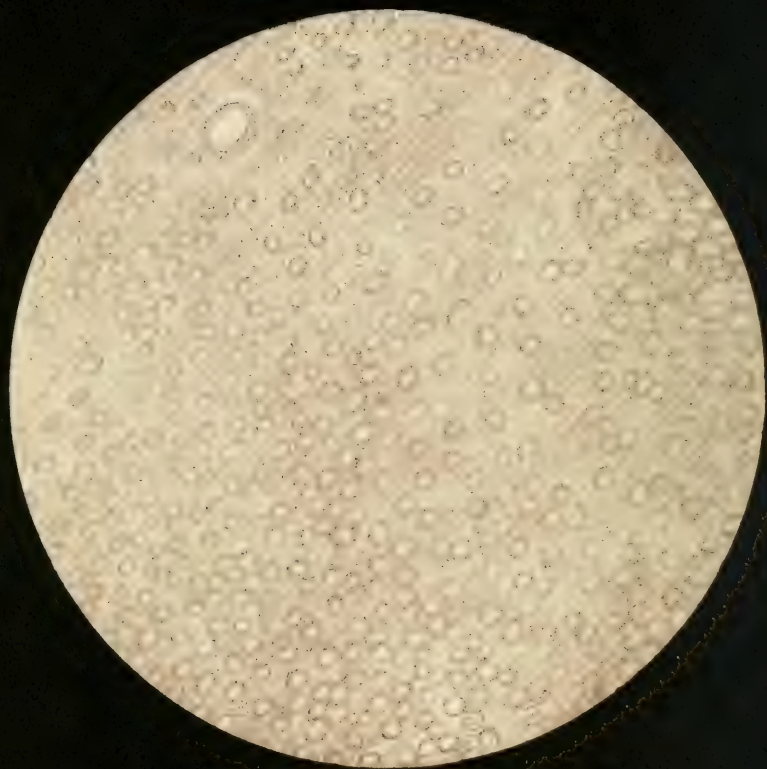
As far, therefore, as our present knowledge carries us, we find first this spasm of vessels; but we know that beyond this abnormal condition there is something on which the spasm depends. It is not unlikely, from the observations of Poincaré and Bonnet, that this lesion is to be found in disease of the cervical, and perhaps other ganglia of the sympathetic.

The recognisable lesions in general paralysis vary according to the intensity of the disease, the time at which death has occurred, and the portions of the nervous system affected. The great disagreement between observers is due to the fact that the post-mortem appearances are sequences of the original lesion, and also that in some cases this, in others that element of the disease is more strongly developed.

The naked-eye appearances are as follows, always remembering, as we must, that in no case can all the lesions mentioned be found :—

1. Great œdema of membranes; adhesions of the closest nature of the pia mater to the subjacent cortex; meningeal apoplexy; degeneration of the cerebral arteries, such degeneration being fatty in only some few cases; thickening and nodulation of the ependyma of the ventricles.

2. Changes in the cortex; greyish-red softening most frequently found in the middle layer of the cortex, and proceeding later to induration. I presume it is this lesion that explains the frequent association of maniacal symptoms with general paralysis. Induration of the most superficial layer, which probably had been previously softened; atrophy of the whole brain;



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induration also of the white matter ; dilatation of the ventricles.

3. Induration and atrophy of the spinal cord, or of spots in it ; on more minute examination, the vessels are seen to be dilated without any thickening of their coats ; much hæmatoidine in the hyaline sheath all over the vessels ; increase of connective tissue and destruction of nervous elements ; the atrophied appearance depends partly on this destruction of the nervous elements, partly on the imperfect nutrition of the brain owing to the diseased vessels ; this increase of the connective tissue is also found in the white substance, either diffused throughout or limited to certain portions.

Plate 15 represents miliary sclerosis in a case of general paralysis. It had affected many portions both of the brain and spinal cord.

Fat granules and granule cells have also been observed in the posterior and lateral columns of the cord in general paralysis, but they seem to bear no relation to the clinical symptoms. They are dependent upon fatty degeneration of the walls of the vessels, and Dr. Huguenin gives them the excellent name of 'the visible form of physiological death.'

Dr. Herbert Major mentions a case in which careful examination showed all the layers of the cells of the cortex healthy, except that they were very pigmental, and that there existed nerve-cells of immense size, situated about midway in the depth of the cortical layer, irregular in contour, with large and numerous branches, these cells being most numerous in the

parietal region, then in the occipital, and most rarely in the frontal lobes. He states also that the neuroglia cells were not so numerous as in brain-wasting.

Calmeil's name will always be so closely associated with general paralysis of the insane, his observations having been the means of attracting the attention of English pathologists to this disease, that a résumé of the post-mortem appearances in some of his cases will not fail to be of interest. He gives 37 cases of this disease, the main lesion being diffuse chronic periencephalitis in a simple state.

In 10 the tissue of the cranial bones was notably red; in 6 the vessels of the cerebral dura mater were injected.

In 15 the cavity of the cerebral arachnoid contained serosity, in 3 false membranes, in 1 coagulated blood.

In 31 the vascular web of the pia mater was red and injected, in 1 infiltrated with pus, in 20 infiltrated with serosity, in 7 thickened, in 9 roughened with opaline lines.

In 35 it adhered more or less intimately to the surface of the cerebral hemispheres.

In 34 the cortical substance of the cerebral hemispheres was red, rose-coloured, or violet, or considerably injected with blood; in 7 it was yellowish, in 1 slate-coloured.

In 14 it seemed to be abnormal by a want of consistence, in 5 by excess of resistance and firmness; in 4 it seemed atrophied. The white substance which constitutes the greater part of the cerebral mass was injected with blood in 23 cases; in 13 it was too firm

and indurated; in 3 too lax and of too little consistence.

In 14 the optic thalami were remarkably red, in 3 yellow or orange; in 7 their consistence was too great, in 1 too little.

In 18 the corpora striata were flesh-coloured or violet, in 4 orange-coloured, in 1 indurated, in 1 too soft.

In 4 the walls of the lateral ventricles were injected, in 5 covered with miliary vesicles, in 6 bathed in serosity, in 2 too soft, in 2 too firm.

In 3 the septum lucidum, fornix, and corpus callosum appeared indurated, in 2 diminished in consistence.

In 9 the pia mater of the cerebellum was red and injected, in 1 slate-coloured; in 12 it adhered to the surface of the cerebellum.

In 17 the cortical substance of the cerebellum was injected at its circumference, in 10 extremely so, in 5 yellow or orange, in 8 diminished in firmness, in 1 too firm.

In 14 the pons was red or rose-coloured in its centre, in 3 it was orange.

In 1 the pons appeared atrophied, in 1 it was too soft, in 1 too firm, in 1 it contained a small cicatrix.

In 11 the medulla oblongata was of a rose or strawberry colour, in 1 of a nut colour.

In 1 it appeared atrophied, in 2 indurated, in 1 too soft.

In 1 its peculiar membrane was surrounded by coagulated fibrine.

In 1 the spinal cord was slender, in 4 indurated, in 1 diminished in firmness, in 4 rose-coloured or injected, in 1 soot-coloured, in 1 surrounded by a plastic coagulation.

The number of brains examined by the microscope was 12. This examination was held in one case in which the delirium presented the characters of mania, in 2 of melancholic delirium, in 1 of ambitious delirium, in 1 of variable delirium, in 3 of dementia with delirious ideas, in 3 of simple dementia, in one where the patient was epileptic.

It is difficult to make a résumé in figures of the number of microscopical alterations which have been noted in these twelve cases of diffuse chronic periencephalitis; nevertheless we may easily be sure that certain alterations are almost constantly found.

Of this number are the serous infiltration of the cortex of cerebral hemispheres, its state of separation, of bloody injection, its mixture with granular elements, either on the walls of the vessels or on the surface of the large nerve-cells, the state of injection of the vessels of the white matter, the development of the corpora striata, the presence of molecular granules and collections of small cells in the midst of the grey matter in the same bodies, the dilatation of the vessels of the cerebellum and of the pons, the formation of granular products on the vessels or in the grey substance of these same regions; finally, the dilatation of the vascular net-work of the pia mater, and its infiltration, either serous or sero-sanguinolent, with the formation of granular elements.

He also gives an account of 45 patients of general paralysis, with chronic diffuse periencephalitis in a state of complication.

Of these, in 13 the diploe of the cranial bones was coloured by hæmatosine and blood.

In 17 the dura mater was injected externally.

In 34 the cavity of the arachnoid contained false membranes or cysts; in 3 the false membranes were vascular; 12 times they were situated in the right cavity of the arachnoid, 9 times in the left.

In 6 the cysts were vascular, in 8 they contained blood, in 5 serosity.

In 20 there was clear serosity in the cavity of the arachnoid, in 7 purulent, in 3 sanguinolent.

In 5 these cavities contained blood, free or scarcely coagulated—once this has been found in the arachnoid surrounding the cerebellum.

In 32 the vascular network of the cerebral pia mater was red and much injected; in 8 it was tinted besides with large suffusions of blood.

In 8 the web of this pia mater was thickened; in 19 infiltrated with serum, in 1 with pus.

In 39 the internal surface of this membrane was as if it were soldered to the cerebral convolutions over a variable, and sometimes considerable, extent.

In 31 the superficial cortical substance was tinted violet, rose, or red; in 4 yellow.

In 10 it seemed softened, in 3 hard, in 4 atrophied.

In 12 it showed one centre, or many, of inflammation.

In 25 the white substance was injected and more or

less tinted by the colouring matter of the blood, in 7 indurated, in 2 somewhat suffused.

In 5 it contained localised inflammatory spots, in 1 pus in cysts.

In 6 the central parts of the brain were found separated. The ventricular walls were often covered with small vesicular projections, causing irregularities on that surface; they were often roughened by large expansions of vessels.

In many cases the cavities of all the principal ventricles contained a certain amount of serum.

In 5 the optic thalami were red, in 1 yellowish.

In 3 they were deficient in consistence, in 1 they contained cicatrices.

In 16 the colour of the corpora striata verged on violet, or a more or less intense red; in 5 on a more or less intense orange yellow; in 3 these bodies were soft, in 2 they contained cicatrices.

In 10 the pia mater of the cerebellum was red and much injected, in 2 reddened by extravasations of blood, in 6 adherent to the nervous substance.

In 23 the grey matter of the cerebellum was coloured violet or red, in 8 it was soft, in 1 too hard.

In 10 the interior of the pons was of a remarkable violet or amaranth tint, in 2 it was deficient in consistence.

In 5 the spinal sinuses were engorged with blood; this blood sometimes formed infiltrations.

In 2 there were deposits of blood outside the spinal dura mater, in 2 beneath it.

In 6 the figure of the spinal cord was red-coloured,

in 5 of little firmness, in 2 too firm, in 1 much injected and atrophied locally, in 4 softened.

Almost always most of the above-mentioned alterations were found united in the same patient.

Thirty-seven brains were examined microscopically.

In 1 these investigations were made on the brain of a patient who had succumbed to an attack of intercurrent congestion of the brain ; in 2 on subjects who showed simple false membranes in the cavity of the arachnoid ; in 1 case on a subject who had cysts filled with blood on the surface of the brain ; in 1 where the cysts were filled with serum. Once these investigations were carried on on purulent fluids, in 4 on interstitial centres in a state of inflammation, once on a spot of central softening, once on a spot of inflammation with induration, twice on inflammatory spots containing cellular fibres in a state of division, once on an inflammatory spot containing the lamellar tissue of cicatrices, twice on cases of softening of the spinal cord.

Finally, the result of all these investigations is that the cerebral capillaries in subjects attacked with diffuse chronic periencephalitis are diseased in various ways ; that abundant extravasations of fibrine are often found ; and that many secondary products are met with, especially in that form of disease that is complicated with phenomena of an apoplectic or convulsive form. I append a case that died of general paralysis after many years' illness.

Man, aged 50. A large quantity of fluid under the dura mater and under the arachnoid, filling up the

space in the skull left vacant by the shrinking of the brain. Brain small and shrunken. Surface of cerebrum very vascular, and convolutions shallow and flat. Surface of cerebellum very pale. There appeared to be a small quantity of grey matter relatively; ventricles very large, containing much fluid. Substance of brain firm. Pons and medulla oblongata on section appeared almost fibrous. Spinal cord remarkably small. In the dorsal and cervical regions it was soft. A great deficiency of grey matter throughout, and the cornua seemed unequal in size and shape.

I cannot close the resumé of the lesions found in general paralysis of the insane without mentioning the conclusions to which Poincaré and Bonnet have come.

1. In general paralysis there is sometimes proliferation of the cellular tissue about the vessels, but it never advances so far as to diminish, still less to completely efface, the calibre of these vessels. Consequently, we cannot attribute the functional and material alterations of the nervous tissue, properly so-called, to a want of blood supply. In one word, there is no sclerosis of brain.

2. The principal and constant alteration of the brain consists in the change of shape and the fatty degeneration of the cells. We find besides, but less frequently, fat globules free in the midst of the granular matter, sometimes isolated, sometimes agglomerated; masses of granulations of a ferruginous tint not surrounded by a common envelope; pigment and hæmotosine in the walls of the vessels and of the fatty granules. Sometimes fatty granulations form vast agglomerations at the circumference of the vessels. We often perceive

enormous globules of fat free or mingled with blood globules. The tubes are always intact.

3. We have found no other modifications in the cord, except a greater abundance of ferruginous granulations in the cells next the ependyma.

4. The cells of the whole chain of the great sympathetic are coloured by brown pigment much more intensely than in other patients, whatever their disease may be. In the ganglia of the cervical regions, and often in the ganglia of the thoracic region, there is evidently substitution of cellular tissue and fat cells for the nerve-cells, which appear relatively much less frequently. Everything leads us to think here is found the anatomical point of origin of the affection, and that the alterations of the brain are only the consequence of the disturbance that this sclerosis carries in its train on the cerebral circulation by paresis of the cervical ganglia. There is always a well-marked pigmentation of the spinal ganglia, and of those connected with the cranial nerves. The fat cells which are substituted for the nerve-cells in the ganglia of the great sympathetic often show a deep colour, that may even be black.

5. All the alterations we have described cause disturbances of nutrition in most of the organs, disturbances which are on the verge of fatty degeneration, or some other modification of their elements; and are manifested physiologically by ataxy at first, and afterwards by enfeeblement of the functions of the life of relation and of vegetative life.

Plate 15, however, shows that sclerosis does exist in the brain in general paralysis. In many cases too in

which no microscopical examination has been made we have records of induration.

Idiocy.—This disease has been divided and subdivided in a manner that only leads to confusion. Dr. Ireland in his excellent article upon it divided it into ten groups: 1, hydrocephalic idiocy; 2, eclamptic idiocy; 3, epileptic idiocy; 4, paralytic idiocy; 5, inflammatory idiocy; 6, traumatic idiocy; 7, microcephalic idiocy; 8, congenital idiocy; 9, cretinism; 10, idiocy by deprivation, that is, by the loss of two or more of the senses. I prefer to follow Griesinger in speaking of two chief varieties, complete idiocy or fatuity, and weakness of mind or imbecility. We may range cretins under both these heads, as the mental power of cretins varies exceedingly.

The objections to this more simple classification are twofold: one, that imbecility is often used as applied to subjects of chronic insanity who have drifted into dementia after acute conditions of disease; and the other, that the gradations in mental power in idiots between fatuity and mere weakness of mind are infinite.

I mean by idiocy a state of mental weakness which has existed from birth or early infancy.

In my first lecture on congenital abnormalities I mentioned several conditions of the brain and membranes with which mental deficiency is necessarily associated. In some few cases, and these exceptional, the configuration of the child is perfect externally, the head well shaped, the limbs well formed, the face in due proportion to the cranium. It is by no means

true that the skull of an idiot is always small. Out of 338 cases, the measurement of which is given by the Massachusetts Commissioners, only 99 had diminished brains. On the other hand, the flat-headed Indians of the Columbia river, whose heads by a mechanical contrivance in infancy have been deformed, so that a depression of the forehead and consequent elongation of the whole head is induced, are not inferior to their round-headed neighbours.

A considerable number of cases seem to owe their origin to falls on the head. One observer alone met with 48 cases in which the idiocy was ascribed to a fall on the head from a height.

Arrest of development, the important lesion in idiocy, may be primarily of the brain, and secondarily of the cranium, or *vice versa*.

We may meet with local thinning of the cranial bones or local thickening; syphilitic exostoses are sometimes seen, and there is one case on record in which with this lesion iodide of potassium cured the child. The thickening of the cranial bones is sometimes very extreme in microcephalous idiots.

I refer you to Griesinger's work for a minute account of the cranial abnormalities in idiots. The main points he insists upon are these: the sutures may close too soon, or unequally at the two sides. From whatever cause they become occluded, there is interruption of the growth of the bones at that spot, narrowing of the cranium at that point, and, when considerable, the influence is extended to parts at a distance. In some cases, however, of premature

ossification of the sutures, corresponding dilatations are found in other parts of the cranium, with the effect of limiting the evil of the contraction, but increasing the deformity. Very seldom are the compensations sufficient for satisfactory and complete cerebral development.

The varieties are these :—

a. True microcephalus would depend upon early closure of the sutures, with early ossification of the base of the skull also, with great interference with the due growth of the brain.

b. If compensation occurs from growth at the base of the brain, we have the Aztec type of idiocy, though in many of these cases idiocy would be a harsh name. There is merely weakness of intellect in certain directions. In several cases that have been examined the cranium was small, the bones thick, and synostosis of the arch had occurred. The basis cranii, on the contrary, was very slightly ossified; the basilar portion almost quite cartilaginous; the petrous portion and ethmoid bone were rather larger than normal; the space for the cerebellum was enormous in all directions.

c. Too narrow crania, shortening of the transverse diameter, caused mainly by early ossification of the sagittal sutures.

d. Too short crania, caused often by early ossification of the lambdoidal suture, generally with compensating development of the region of the anterior fontanelle; or by extended ossification of the frontal with the parietal bones.

e. Too low crania, caused by ossification of the

wings of the sphenoid with the frontal bone and the squamous portion of the temporal bone.

f. Unsymmetrical obliquely narrow crania, caused by unilateral ossification, either of one half of the coronal suture, or of one half of the lambdoidal suture, with compensating extension towards the opposite side.

9. The sinosto-basilar form ; cretinism in the proper sense of the term, though cretins exist without this abnormality. This form is due to premature ossification of the base, ossification of the synchondrosis between the sphenoid and the basilar portion, which in the normal state does not occur until the fifteenth year, and sometimes not till the twentieth year. This shortening of the base of the cranium causes a great curvature upwards of the base of the cranium, a small angle at the point of union between the sphenoid and the basilar portion, and a steep clivus, with a condition of face that is called prognathism. Dr. Stahl, finding this abnormality frequent in cretinism, examined the condition of the clivus. In 104 insane patients, he found it only in three, one of whom had previously been imbecile.

When we proceed to a resumé of the lesions of the brain, we meet with a greater variety of conditions.

1. The brain may be symmetrical, and simply very small.

2. The arrest of development may be shown not only in a small size of brain, which, however, may be symmetrical, but in deficiency of the grey matter of the convolutions. Thus, Dr. Mierzeiinsky mentions an idiot,

fifty years of age, who had had the capacity of a child a year and a half old. The size of the head was about that of a child of one year old. The encephalon weighed 369 grammes; the cerebellum, pons, and medulla oblongata were about the usual size, the deficiency being in the cerebrum; the corpus callosum was only one-third of the usual length; the convolutions were simple and undeveloped, the grey matter deficient.

3. Inequality in the size of the two hemispheres, owing to atrophy of the smaller; this atrophy either from arrest of development due to stenosis of that side of the cranium, or from sclerosis, the sequence of some form of encephalitis. This encephalitis may have existed during foetal life, or in the early months of infancy. The sclerosed portion of brain is often also pigmental. Atrophy sometimes occurs from sclerosis round an old apoplectic clot.

4. A want of symmetry of the cerebellum, the pons, and the medulla oblongata, sometimes existing alone, sometimes in connection with a want of symmetry of the brain proper. This abnormality of the pons and the medulla oblongata is often found to depend on the spheno-basilar synostosis before mentioned.

5. A stunting of both anterior lobes with defective development of the olfactory bulbs, or of the posterior lobes. Examples of the former abnormality have been reported by Dr. Cramer of Soleure. He has shown the brains of three microcephalous cases. The dimension of the smallest amounted to 360 cubic centimetres. In all these brains the island of Reil was left uncovered, owing to the defective development of the frontal and

parietal gyri. In the case of an imbecile woman, reported by Dr. T. H. Simon, the convolutions were more simple than usual, and those of the island of Reil were replaced by a smooth layer of grey matter.

6. Absence of the upper convolutions of the brain, disclosing the ventricle; this absence of the convolutions being generally rather a result of disease than an arrest of development.

7. Congenital or early hydrocephalus of chronic form; sometimes the sole lesion, and accompanied by great thickening of the ependyma, sometimes only secondary to the want of development in the brain. The brain in these cases is often abnormally small.

8. An abnormal amount of the grey matter in proportion to the white, or an unusual distribution of the grey matter in regions where it generally has no place.

9. Absence or deficiency of many of the special parts of the encephalon. Thus, the cerebellum may be absent, or the pineal gland. The fornix may have a very deficient formation, or the olivary bodies, the crura cerebri, the corpora mamillaria, the optic thalami, or the corpora striata. There may be considerable bilateral atrophy of the optic or the auditory nerves, or more commonly this may be unilateral; and lastly, the corpus callosum is sometimes absent, or only represented by a very thin band of fibres; and this latter abnormality is found to be accompanied with some weakness of intellect rather than complete idiocy.

10. In a few cases there is found a peculiar condition, the presence of a fifth ventricle in the olives of the cerebellum, a condition that exists in the brain of birds.

11. Lastly, various abnormalities of the pituitary body. This brief resumé of the lesions of idiocy would be incomplete, unless we mention the occasional abnormal narrowness of some of the cerebral arteries from contraction of the foramina of the skull. In many idiots, not only is there much deformity of face, but of the whole body. They are frequently dwarfed, with a thickness and heaviness of all the limbs. In cretins the cerebral weakness is constantly accompanied by goitre, and a great fulness of the throat is to be found in many idiots. I have now under my care a man of nearly 40 years of age, with the mental capacity of a child of five, very sweet-tempered and religious. In him the fulness of the throat amounts distinctly to goitre, and occasions him some difficulty in deglutition.

Slowness in the development of the teeth is also seen in cretins and in many idiots, and in both the teeth are apt to decay early. A keel-shaped palate is very common in congenital idiocy. Cleft palate is not very unusual.

The development of the sexual organs is often arrested in idiots. In advanced cases of cretinism, and in many complete idiots, the organs are quite unfit for generation.

In semi-cretinism unfortunately this function is not wholly lost, and the disease may be transmitted from parent to child.

But recent pathological research, and still more the results of treatment in modern times, have shown that the state of mental deficiency in idiots bears no definite relation to the cerebral lesion. We can form no

reasonable prognosis on these cases without taking into consideration the medium in which they live, including the food, the air, the comforts of life, and especially the moral and intellectual surroundings.

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LECTURE IX.

APHASIA.

FOR the expression of ideas in speech there are three requisites: 1. The memory of words. 2. The remembrance of how to say them. 3. A proper condition of health of the external organs of voice and articulation with their muscles and nerves. A want of action, however, in these latter, though it would prevent speech, would not be aphasia.

Aphasia is of three kinds: 1. Amnestic or amnesic aphasia, the loss of memory of words. 2. The more common form, the loss of memory of how to say words. The seat in the brain of the first faculty, the memory of words, has never, I think, been quite made out. It may exist in many parts of the brain, as well as in Broca's spot. The seat of the second faculty, the remembrance of how to say words, has been located by Broca and others in a particular spot in the brain. 3. The loss of speech consequent on the third form of aphasia is due to the want of co-ordinating power over the muscles of articulation.

Dr. Bartholow says the existence of a distinct centre of the faculty of language, although not absolutely proven, a number of facts render exceedingly

probable. The precise limitation to the third left frontal convolution attempted by Broca, or to the walls of the left sylvian fissure as suggested by Meynert, may not, in the present state of our knowledge, be considered anything more than plausible conjecture. It were better, probably, to hold with Hughlings Jackson, that the district of cerebral matter supplied by the left middle cerebral artery is most intimately concerned in the function of language; for this will include all that region of the anterior and middle lobes which has from the time of Gall been supposed to be the seat of this faculty. It is obvious, he says, that three forms of loss of the language faculty may exist: Amnesic aphasia, paralytic aphasia, ataxic aphasia. In amnesic aphasia, the defect must be strictly limited to the vesicular matter in which the memory for words is situated. All other forms of expression may be retained, the mental conception may be accurately defined, but the store of words for giving vocal form to the ideas has been obliterated. It is rare indeed to find a perfect case of amnesic aphasia, for the destruction of the one faculty is usually associated with more or less impairment of the others. The cases of paralytic aphasia are very numerous. These arise from embolism of the middle cerebral artery, apoplectic extravasations in the white matter and in the corpus striatum, and tumours, such as aneurism, glioma, gummata, and other forms. The very common association of right hemiplegia and aphasia is now perfectly well known. In these cases an interruption exists in the communication between

the language centre and the vocal apparatus. In the ataxic form of aphasia there is no disorder of the language centre, nor in the transmission of the motor impulse through the motor channels to the olivary bodies; but owing to disease of this part of the cord, the muscles concerned in the production of articulate language cannot be combined to execute the varied movements necessary. As Schroeder van der Kolk supposed that the olivary bodies were intimately concerned in the function of spoken language, he has been interpreted to mean that this is the real language centre. Undoubtedly he did not refer the idea or mental conception and the memory of words to the olivary bodies, but only the mechanism of vocal expression.

Broca finds, and his views and observations have been confirmed in the main (with the exceptions to be mentioned further on), that the seat of lesion in cases of aphasia is the posterior part of the third left frontal convolution. In Broca's description of this portion of the brain, the third frontal convolution presents a superior or internal border adjoining the tortuous border of the middle convolution, and an inferior or external border, the relations of which differ according as they are examined before and behind. In its anterior half this border is in contact with the external border of the most external orbital convolution. In its posterior half, on the contrary, it is free, and separated from the temporo-sphenoidal lobe by the fissure of Sylvius, of which it forms the superior border. The inferior border of this fissure is formed by the

superior convolution of the temporo-sphenoidal lobe. When the two marginal convolutions, the superior and the inferior, are drawn away from the fissure of Sylvius, there appears a large and slightly prominent eminence, from the summit of which five small, simple convolutions, or rather five straight folds, radiate in a fan-like manner. It is the lobe of the insula, which covers the extra-ventricular nucleus of the corpus striatum, and which, rising from the bottom of the fissure of Sylvius, is found to be structurally continuous, by its cortical layer, with the deepest or most deeply-seated part of the two marginal convolutions above-named (forming the superior and inferior borders of the fissure), and by its medullary layer with the extra-ventricular layer of the corpus striatum. The result of these structural relations is, that a lesion which propagates itself continuously from the frontal lobe to the temporo-sphenoidal lobe, or *vice versâ*, will pass almost necessarily by the insula, and that from thence it will most probably extend to the extra-ventricular nucleus of the corpus striatum, since the proper substance of the insula, which separates this nucleus from the surface of the brain, forms only a very thin layer.

In considering, however, the close relation of the corpus striatum with this third frontal convolution, and the important part it plays in being what Dr. Broadbent calls 'a way out for words,' it is necessary to remember that it is not quite the only way out. The fibres of the corpus callosum apparently connect symmetrically corresponding convolutions of the two

hemispheres; and it is far from improbable that in lesion of the left corpus striatum the right corpus striatum may form the channel for the transmission outwards of words. These few preliminary observations were necessary before the pathological anatomy of this disease was entered upon.

In a very large proportion of cases of aphasia, the loss of speech has coincided with right hemiplegia. This is not only the case when lesion has occurred after birth, but in the few cases on record of congenital aphasia the right limbs have suffered.

Thus Waldenburg reports a case of a child in whom, shortly after birth, the parents observed partial paralysis and wasting of the right side of the body. As it grew older, and up to six years, the child had given little evidence of speech. He was not deaf, and was quite intelligent. The child, with only slight interference with the motion of the tongue, did not even try to bring forth articulate sounds. The case is the more interesting in that the mother, when three months pregnant with this child, was seized with right hemiplegia and loss of speech. She improved slowly, but six years afterwards, when first seen by Dr. Waldenburg, there remained a certain weakness in the right extremities, and her speech was not only somewhat thick, but she had difficulty in expressing herself properly. Dr. Waldenburg believed the child's condition to be congenital, caused by an intra-uterine affection. Here the left hemisphere was affected before the child had arrived at the natural age for commencing to speak, but the right or healthy hemisphere did not meet the want.

The cases naturally divide themselves into two classes of lesions : one of Broca's spot, and the other of the way out from it towards the nerves used in articulation. Theory is of very little use in determining the question. It is only by numerous observations of symptoms and of the exact seat of lesion that we can arrive at any intelligent understanding of the matter.

Now the kinds of lesions met with are manifold :

Thus Case 1. Right hemiplegia. Aphasia. A little thickening of the pia mater and arachnoid over the convex surface of cerebrum. A spot of softening, the size of half-a-crown, exactly in Broca's spot.

Case 2. Girl, aged 13. Right hemiplegia. Aphasia. A little discolouration at the posterior portion of the third left frontal convolution. Much black discolouration along the fissure of Sylvius on the left side. Beneath this latter region was an old clot, partially discoloured, with creamy tissue all round it, extending slightly into the anterior lobe, but mostly into the middle lobe, and implicating the left corpus striatum, except the upper layer of it, which forms part of the floor of the ventricle. This clot was the result of a ruptured middle cerebral artery. Just at the commencement of the fissure of Sylvius the artery was enlarged to the size of a small haricot bean. This aneurism had given way. No embolism. The lesion immediately causing death was the rupture of a small vessel in the left lateral ventricle, on the outer and upper side of it, just above the corpus striatum. The whole of the left lateral ventricle was full of clot, which

filled all the cornua, and had found its way, to a small extent, into the right lateral ventricle. The posterior portion of the third left frontal convolution at the seat of discolouration was stained to about a fifth of an inch in depth, and sensibly softened over a space the size of a walnut.

Several of Dr. Hughlings Jackson's cases were living when he wrote his paper in the London Hosp. Reports on aphasia, and we are deprived, therefore, of the accuracy of post-mortem records. From the manner in which the morbid phenomena occurred, and their coincidence with valvular disease, or some other abnormality of the heart, there seems no doubt that the lesion was embolism. In his Case 10, death occurred from rupture of an aneurism of the middle cerebral artery. In his Cases 27 and 28, syphilis seems to have given rise to the lesion. Several others of his cases were probably due either to small hæmorrhages or to very localised softenings.

Darx recorded 140 cases of disease of the left hemisphere, accompanied with aphasia and right hemiplegia, and concluded that the faculty of language must be connected with the left hemisphere.

Trousseau so far corroborated this experience by finding in 134 cases of aphasia disease of the left hemisphere in 124, while 10 cases were contradictory of Darx.

Darx, Junior, placed the organ of the manifestations of thought by speech at the point of union of the middle with the frontal lobe of the left hemisphere.

In his clinical lectures, Trousseau mentions a case of

aphasia and right hemiplegia in which there was found at the autopsy yellow softening on the left side of the lower marginal convolution of the lower portion of the transverse parietal convolution, and of the convolutions of the insula. At first sight, the frontal lobe seemed to have escaped; but on drawing away the edges of the Sylvian fissure, the softening was seen to extend from the convolutions of the insula to the lower portion of the transverse frontal convolution; and, moreover, the third frontal convolution was itself softened in its posterior portion, that is, in the part nearest the sulcus of Rolando.

Cases as definite as this are given by M. Broca himself, and can be added to by most hospital physicians. Dr. Charcot exhibited to the members of the Biological Society several brains removed from old women who had died in the Salpêtrière, and who had, for a variable period during life, suffered from loss of speech. In most instances the lesion was complex, as in Broca's first case. Thus there had generally been paralysis as well as aphasia, and the second and third frontal convolutions were not the only spots where there was softening or hæmorrhage; but the lobule of the insula and temporo-sphenoidal lobe were also the seats of an anatomical lesion, which had probably occurred simultaneously everywhere, or which had resulted from the extension of softening by continuity of tissue.

In Broca's second case, in which there was aphasia without paralysis of limbs, and the patient had suddenly lost his speech since a fit of apoplexy which he had eighteen months before his death, the posterior third

of the second and third left frontal convolutions was alone destroyed, over a space of about 15 or 18 millimeters. The transverse frontal convolution was normal, whilst inferiorly the lesion extended as far as the lobule of the insula, but without involving it. The result of this loss of substance was a cavity full of serosity, and closed externally by the pia mater. The walls of this cavity were firm, and on them were small spots of an orange-yellow colour, proved afterwards to be of a blood origin. This was, therefore, an old hæmorrhagic cyst.

I will not take up your time by multiplying instances of this nature. Some are recorded in St. George's Hosp. Reports, Vol. 2. They are numerous, and would be conclusive if there were no exceptions. Still, in recording the pathological anatomy of aphasia, it is necessary to note the exceptional cases also.

These are of two kinds: (1) those instances of aphasia occurring with left hemiplegia; and (2) grave lesions of Broca's spot without loss of speech. To these may be added also those instances of aphasia with lesion on the left side, but of some other portion of the brain, and not of Broca's spot.

Of the first kind of exceptions there are not many examples on record. Let us take a few in order:—

1. Trousseau mentions the case of a patient, named Marcou, affected with hemiplegia and aphasia. The lesion was probably of a syphilitic nature, as the patient improved in all respects very much under mercury and iodide of potassium.

2. Case 18 of Dr. Hughlings Jackson:

Mrs. B., aged 32, after a natural labour, was sud-

denly seized with complete paralysis of the left side, and was perfectly unable to talk. Of course, it is possible in this case that there was disease on each side of the brain, but the left hemiplegia and the aphasia occurring simultaneously and instantaneously renders the idea of a double lesion difficult.

3. Case 19 of Dr. Hughlings Jackson:

Man, aged 49, suddenly complained of severe headache, and left his work at 7 P.M.; after a walk he sat down and became speechless, and by the time he was got to bed he had lost the use of the left arm and leg. He did not speak at all for six weeks, except to say 'Yes' or 'No.'

4. Case 38 of Dr. Hughlings Jackson:

A gentleman, who fourteen years before had a bad fall from his horse, was suddenly taken with loss of consciousness without convulsion. The result of this fit was entire paralysis of the left side, and for eighteen months total loss of speech. There was ptosis of the left eyelid. He lingered for five years and a half, the paralysis continuing up to the time of his death. After the first eighteen months he seemed to be recovering his words and could enunciate parts of them only, but these in no way corresponded to his seeming intentions; his ideas being imperfect, and his speech both confused and ataxic. For a little while there was paralysis of the bladder; but the tone of that viscus soon returned. There was no autopsy.

Besides these, there are a few more cases on record, scarcely a dozen in all, in some of which there is no post-mortem record.

Dr. W. A. F. Browne speaks of the numerous cases of left hemiplegia with aphasia, of which not less than six have been noticed in this hospital (the Crichton Royal Institution). He states that Baillarger, in like manner, records 155 cases of hemiplegia, of which only ten were upon the left side, from which it would appear that in aphasia right is to left hemiplegia at 15 to 1. I believe the proportion of right hemiplegia to left in aphasia is enormously larger than this where the aphasia and the hemiplegia were coincident in point of time, and not due to lesions occurring at different periods.

The second class of exceptions includes those in which there was lesion of the posterior portion of the third left frontal convolution, but no loss of speech.

Case 1. Not a satisfactory one, reported by Professor Velpeau :

The patient, a hair-dresser, was admitted into the Charité Hospital, suffering from incontinence of urine. He was an extremely tiresome talker, and died three weeks after admission, without having presented any symptom of cerebral disease, any difficulty of articulation, or defect of speech. On dissection there was found hypertrophy of the prostate, stricture of the urethra, and old disease of the mucous membrane of the bladder. The dura mater was found to be firmly adherent to the mass of the brain. The right anterior lobe of the brain was completely destroyed by a voluminous tumour having all the characters of scirrhus. The left frontal lobe also had been encroached upon, and was to a great extent destroyed. This description is not sufficiently exact to enable us to form an opinion as to whether Broca's spot was much injured.

Case 2. The next case was observed by M. Aug. Bérard:

The patient, a miner, was injured by the explosion of a mine. He did not lose consciousness; managed to creep out of his hole, and asked to be taken to M. Bérard's house. The whole frontal region was laid open, the integuments hung in shreds, the bones were splintered and in detached fragments, and the brain was exposed. Both anterior cerebral lobes were completely destroyed, and in their stead was a mixture of blood, of bony splinters, and of brain substance. In spite of this frightful accident the man could relate, in all its details, how the accident had occurred.

Case 3. Dr. Batty Tuke's and Dr. Fraser's case:

The history of this case is that of an apoplectic seizure eleven years previous to the date of observation. Her friends affirm that she was insensible for some weeks. No amount of cross-examination of her sister, with whom she resided at this time, could elicit any evidence of paralysis; there was no distortion of the face, no lameness or want of power in either hand, but complete speechlessness existed for some time. From this, however, she must have totally recovered, for previous to her admission she seems to have been rather talkative. She died of paraplegia consequent on atrophy of cord, with caries of the vertebræ. On stripping off the dura mater on the left side, some slight adhesions were found between the layers of the arachnoid. These were easily detached, and exposed an excavation of the brain substance at the postero-infero-external part of the left frontal lobe. Its outline was

irregular, its cavity filled with serum, and narrow white bands sprang from its sides. The serum was opalescent, but otherwise normal, and it was held in by the visceral arachnoid. On emptying the cavity, its dimensions were found to be as follows: In its long axis, from before backwards, parallel to the fissure of Sylvius, two and a quarter inches obliquely, vertically one and three-sixteenths of an inch; in its deepest part it was three-quarters of an inch, but generally only half an inch. This lesion had destroyed posteriorly the inferior fourth of the ascending parietal convolution, leaving a small posterior portion of the knuckle in which this gyrus ends, the inferior third of the ascending frontal, the inferior margin of the second frontal, and the posterior half of the third frontal convolution. At the inferior margin there was a narrow ridge of slight eminence, which might have been the remains of the inferior border of the third. With these exceptions, the destruction of the posterior half of this convolution was complete, both as regards its grey and white matter. Its inferior boundary was the superior marginal convolution. The bottom seemed to be an anatomical limitation, as it was smooth, rounded, and presented no evidence of morbid action. Incision proved it to be the extra-ventricular nucleus of the corpus striatum. The edges of this lesion, implicating the convolutions, were ragged, which was suggestive of erosion; but there was no indication, by induration, softening, or thickening of the membranes, of inflammatory action. Microscopical appearances: chiefly those of fatty degeneration and amyloid bodies. It is

true that this case was objected to by Dr. Wilks, on the ground that the patient did not use nouns correctly. The woman had the faculty of speech intact, but had forgotten certain words.

Case 4. My own case :

Henry E. had been under treatment for some time for syphilitic node on the left side of the forehead. It disappeared to a great extent under treatment, and he left the Bristol Infirmary, but was readmitted in a few weeks with very distressing pain in the left side of the head anteriorly. He died comatose, but was able to talk to me within a few hours of his death ; and he had not the slightest loss of speech all through. Coma, lasting only a few hours, came on rather suddenly. The frontal bone was somewhat thickened just above and to the left of the left orbit. Beneath this spot the dura mater was firmly adherent to the bone, and also to the parts beneath it, so that it could scarcely be separated from the other membranes of the brain without tearing them. The brain at this spot was hard, and felt as though a hard tumour was contained in its substance. It was yellowish externally, and was so placed that the lower portions of all the frontal convolutions on the outside of the anterior lobe of the left hemisphere were implicated in the mass. On section, the mass was found to be hard and of a yellowish-white colour, surrounded by congested vessels. It extended to the posterior border of the anterior lobe, and about half an inch into the middle lobe, and was the size of a small hen's egg. Between the two portions of the tumour in the anterior and

middle lobes the artery was compressed, so that its canal was almost wholly blocked up and rendered useless. Almost all the central white substance of the middle lobe and of the left corpus striatum was diffuent. A little clear fluid was found in the lateral ventricles.

In connection with this particular point, a case recorded by Dr. Bristowe is important, as showing that the corpus striatum is not the only way out from the third convolution. The left corpus striatum was entirely destroyed, but the power of speech had returned in three or four weeks after the apoplectic attack.

The third class of exceptions will include those in which aphasia has occurred, and some other portion of the brain besides either the left or the right third frontal convolution has been the seat of lesion.

1. Dr. Charcot's case. Woman, aged 49.

She had become hemiplegic on the right side, and aphasic since an apoplectic fit which she had had eight months previously. Intellect and memory seemed to be preserved, but her power of articulate language was restricted to the utterance of the monosyllable 'ta,' which she habitually repeated, with very great rapidity and distinctly, four or five times in succession, whenever she attempted to answer a question or to communicate her own ideas. The tongue moved freely and in all directions. An examination of the brain showed that there was softening (1) of the so-called lower marginal convolution in all its extent, and of a portion only of

the second temporal convolution of the temporal lobe ; (2) of the lower extremity of the two posterior convolutions of the insula of Reil. In depth, the softening extended in the direction of the corpus striatum ; the whole of the extra-ventricular nucleus and the posterior half of the intra-ventricular nucleus were also softened. The thalamus opticus was normal. The transverse parietal and transverse frontal convolutions, the three antero-posterior frontal convolutions were examined thoroughly, one after another, with the greatest care, in M. Broca's presence. Even microscopically Dr. Charcot and M. Broca considered that there was no lesion in them.

Case 2. Dr. Vulpian's case. Woman, aged 73. Aphasia, and later on right hemiplegia.

Dr. Vulpian says : ' Dissection did not disclose the lesion, which I fully expected. I found a broad patch of softening of apparently recent date in the posterior half of the white supra-ventricular nucleus of the left cerebral hemisphere, and no trace of disease in the frontal or other convolutions. Old lesions, slight in degree, lacunæ, were seen in the corpus striatum and optic thalamus on the same side, and an analogous lesion, of still smaller extent, but of as old a date, in the right corpus striatum. Both middle cerebral arteries were very atheromatous ; but whilst the right artery was still pervious to the blood, the left one was completely or almost completely plugged up in two places, separated by an interval of about one centimetre from one another, owing partly to the atheromatous thickening of its walls, and partly to an indurated

fibrinous deposit of manifestly old date. This deposit seemed to have been the result of thrombosis rather than of embolism.

Cruveilhier has brought forward several curious instances in which the loss of speech was a prominent symptom, while the disease was not found in the anterior lobe, but in some other part of the brain.

Andral has recorded 37 cases of cerebral hæmorrhage, observed by himself or by others, in which, whilst the morbid condition occupied one or both of the anterior lobes, the power of speech was affected 21 times and unaffected 16 times. On the other hand, he has collected 14 cases in which the power of speech was lost, yet no alteration had taken place in the anterior lobes. In 7 of these 14 cases the lesion was situated in the middle lobes, and in the other 7 in the posterior lobes of the brain.

Case 3. Man, aged 48. Admitted into the Bristol Infirmary, and died the same night. The wife states that eighteen months ago he lost his speech altogether for four months, without any paralysis of the limbs. Nine months ago, and therefore five months after the recovery of his speech, he had loss of motion and sensation on the right side, with some muffling of speech, but no aphasia, and never recovered from this condition. He died in a third attack. A clot of considerable size occupied the middle portion of the right hemisphere, reaching from the external border of the right corpus striatum to the lateral surface of the hemisphere, through which the clot protruded on the slightest pressure. The cerebral substance around the clot was

all broken up and creamy. The right lateral ventricle itself healthy. The left optic thalamus was partially destroyed, its seat being occupied by a depression stained yellow, the floor of which was very soft. This condition extended some way also into the crus cerebri of that side. This was evidently the seat and formed the remains of an old clot. Broca's spot was perfectly healthy. It certainly was no softer than the other healthy parts of the brain. There was no lesion that accounted for the first loss of speech for 4 months. Cerebral vessels fairly healthy, and the rest of the cranial contents. Small granular kidneys.

Case 4. Man, aged 30. Syphilitic. Aphasia and right hemiplegia for six weeks. Admitted into Bristol Infirmary, March 1873. Broca's spot and every part of both hemispheres quite healthy. Pons, the seat of softening, the size of a filbert, occupying rather more of the left half than the right, but passing a good deal over the middle line. The nervous tissue were here quite deliquescent. Vessels of the circle of Willis a little fatty. Both these latter cases may have been instances of lesion of parts concerned in the way out from the anterior portion of the brain.

It is difficult, if not impossible, to reconcile all these exceptional cases with Broca's law. We must either admit that the seat of the faculty of speech is located in several portions of the brain, or that lesions of other parts cause paresis of the faculty either by pressure or by reflex irritation, or at least that a corresponding portion of the right anterior lobe is or may be the seat of this faculty. Nor do the pathological observations,

which show that lesion of the corresponding spot in the right hemisphere has not been accompanied by aphasia, militate entirely against this latter view. It is recognised pretty generally that right-handedness depends on the earlier and more complete nutrition of the left side of the brain. Even if Gratiolet's statement is incorrect, that the anterior convolutions of the left side are earlier developed than those of the right, yet, at any rate, according to Broca, in forty brains the left frontal lobe was found to be on an average heavier than the right; and a similar assertion with respect to the weight of the entire left half of the cerebrum has been made on the basis of very numerous observations by Dr. Boyd.

This more complete nutrition is due partly to the somewhat greater size of the left carotid, mainly, probably, to the fact that the blood enters the brain on the left side much more directly than on the right; that in fact it has not the temporary obstruction to contend with that it has on the right side in its course from the aorta to the innominate, and from the innominate to the carotid.

It is only reasonable to believe that in the case of left-handed persons the ordinary nutrition of the two sides of the brain has been reversed, and that the right side has been better and earlier nourished. In a very interesting paper on left-handedness Dr. Pye Smith calls attention to two cases of aphasia occurring in left-handed persons, recorded by Dr. Hughlings Jackson and Dr. Ogle. In both these patients there was paralysis of the left side; so that it seems likely that in these two left-handed people the right half of the brain

had the functions, if not the structure, which ordinarily belongs to the left. In Dr. Wadham's case also a partially left-handed lad was attacked with left hemiplegia and loss of speech; he had partly recovered at the time of his death twelve months later, and then the right insula and adjacent parts were found softened. Bearing on this subject it is well to notice that in two brains of left-handed persons examined by Dr. Broadbent and Dr. William Ogle the ordinary conditions of the two hemispheres were reversed, the greater complexity of convolution occurring in both on the right side, and not on the left; and there is no reasonable doubt but that right-handedness and left-handedness are associated with more highly developed frontal convolutions, in the one case on the left, in the other on the right side.

It may well be that Broca's spot and the corresponding portion of the right hemisphere are seats of the faculty of speech; that the spot on the left side of the brain, being more highly nourished, either takes in the whole faculty, there being no need in most cases for the right side to be called into use, or that the left side may be the seat of the higher faculty, the right of the lower; that, in other words, and those Dr. Hughlings Jackson's, the left is the leading side of the brain, the right the automatic. Dr. Jackson believes that the involuntary utterances of aphasic patients are the result of action of the right side.

What I have said, however, may give you some idea of the pathological anatomy of aphasia, whatever may be the true pathology.

To sum up in a few words :

1. The posterior portion of the third left frontal convolution is the main seat for the faculty of speech.

2. That it is not the only seat seems proved by three classes of exceptional cases, viz., those in which aphasia has occurred coincidently and synchronously with left hemiplegia; those in which Broca's spot has been the seat of grave lesion without aphasia; and those in which aphasia has occurred without lesion of Broca's spot, but with lesion of some other portion of either left or right hemisphere.

3. Some of the instances of aphasia with left hemiplegia have been met with in left-handed people, and therefore in persons with the right anterior lobes presumably or certainly developed with a complexity of convolutions that is generally only found on the left side of the brain.

4. To use Dr. Broadbent's words, 'The left third frontal gyrus being the outlet for expression, the left corpus striatum necessarily takes the lead in the production of spoken words; but a way round exists, probably from the left to the right third frontal gyrus, by the corpus callosum, and thence to the right corpus striatum. Thus speech, though temporarily embarrassed by damage to the left corpus striatum, is recovered; whereas if the cortex of the left third frontal convolution is damaged, or its fibres, both to the corpus striatum and to the corpus callosum, cut through, speech, having no other outlet, is lost.'

Glosso-Laryngeal Paralysis.—In uncomplicated

cases the disease is manifested by paralysis of swallowing, of speech, of phonation. The lips, the tongue, the larynx, the pharynx, are all more or less paralysed. There is paralysis of all the parts employed in talking. There seems to be a great tendency in the paralysis to become general; but at present we will confine ourselves to the uncomplicated disease.

Deglutition is difficult, the patient being only able to take liquids; certain words are especially difficult; the patient cannot pronounce the vowels o and u, nor the consonants p, b, m, n, k, c, t, which require the intervention of the tongue and lips. This condition is different from the mumbling articulation of cerebral paralysis with partial implication of the facial nerve; still more does it differ from the loss of memory of words, or of the power of articulating them, we meet with in aphasia. The voice is often either feeble or absolutely whispering, a phenomenon depending on lesion of the spinal accessory nerve. This feebleness of voice also is due in some cases to weakness of expiratory effort, when the spinal roots from which the phrenic nerve derives its nervous influence are coincidentally affected. The saliva constantly dribbles from the mouth, as the patient is unable to retain it by means of the lips. This flow of saliva is not only due to the labial paralysis and loss of power of swallowing. The quantity and quality of the saliva are altered, as a direct result of the disorder of innervation. Schulz has shown in one case that galvanization of the facial nerve has caused the disappearance of the abnormal secretion.

The food collects between the cheeks and the

teeth. The muscles of the soft palate hang down, and if the patient opens the mouth and takes a deep inspiration the palate is not elevated as it is in health, and the posterior nares cannot be closed.

The post-mortem examinations on such cases have been very few. The disease is rare in itself, and often so gradual in its progress that cases will not remain in hospital until the fatal event occurs. Still, there are some few on record, and it is necessary to call your attention to them. It is remarkable that of those few the disease is, in several instances, associated with progressive muscular atrophy. It will be my duty to call your attention to this connection by-and-by.

In the first of Trousseau's cases the post-mortem examination was entirely negative as regards the anatomical lesion; perhaps, as he himself says, because it was incomplete.

In the second case there was found very marked atrophy of the roots of the hypoglossal nerve, without any alteration of the muscular fibres themselves. The medulla oblongata was apparently also of greater consistence than normal.

In Trousseau's third case there was found well-marked thickening and grey discoloration of the dura mater on a level with the medulla oblongata, and as far down as the roots of the fourth cervical pair. This thickening was due to a considerable increase in the amount of fibres of connective and fibro-elastic tissue, and seemed to result from a chronic congestive process, as shown by the great number of capillaries and of deposits of hæmatine external to them. The roots

of the hypoglossal and spinal accessory nerves were atrophied, and reduced in several places to the neurilemma; and at the spot where the spinal accessory was in contact with the dura mater there was adhesion of the neurilemma to the fibrous envelope of the cord, and a deposit of a nucleus of connective tissue the size of a pea. A good many motor roots in the cervical region were thinner than natural, from partial disappearance of the nerve-tubes. With the aid of the microscope, the neurilemma was seen to preponderate everywhere over the nerve-tissue properly so-called, and notable hyperæmia could be detected everywhere, together with greyish discoloration of the neurilemma. The cord itself, at the upper part of the anterior columns, was as congested and of the same colour as the posterior columns are found to be in cases of progressive locomotor ataxy. The fibres of the palsied muscles of the tongue, soft palate, lips, chin, and cheek were unaltered. There was diminution of size and incipient fatty degeneration of some of the muscles of the right leg, especially of the peroneus longus, tibialis anticus, and quadriceps femoris. Here, as in the two following cases, the special lesion was complicated with progressive muscular atrophy.

Dr. Duménil's case showed atrophy of the hypoglossal, facial, and spinal accessory nerves; complete motor paralysis of the tongue, incomplete of the face; integrity of the muscles of the tongue and the face; atrophy of the anterior spinal roots; incomplete paralysis of the limbs; incipient muscular atrophy.

Dr. Wilks' case showed no nodes or other signs of

disease of the cranial bones. The brain was tough and hard. There were no signs of formative disease : the changes required to be looked closely for. But on opening up the visceral arachnoid, there was a most obvious atrophy of the roots of the hypoglossal nerve, which had quite lost the natural white opaque appearance of nerves, and were thin little gelatinous threads as they crossed the corpora olivaria. In the same condition were the inner roots of the spinal accessory, and also very markedly the whole of the anterior roots of the spinal nerves, especially the cervical ; and the sacral least. The anterior view of the cord was remarkable ; the outer aspect was flat, not round ; yet, it was harder than natural, so that mere flaccidness was not the cause of this ; the anterior roots also came from a line nearer the middle line than is natural. On section, the anterior half of the white matter was atrophied ; it was white, harder than natural, and on the section it stood out, while the grey matter receded ; the latter was larger than natural, it was darker, containing obvious vessels, and at the lower part of the cervical cord it was double the natural size, and showed a red colour finely mingled with yellowish white ; this part, so affected, was not of great length ; generally the redness and largeness of the grey part, and the thin hard shell or coat-like layer of white matter, made the pathological state of the spinal cord. In the medulla oblongata, as seen from the front, nothing diseased was visible, except the state of the nerve-roots, as before stated. But opening up the arachnoid over the fourth ventricle, and drawing down the

medulla oblongata to look at the fourth ventricle, there was a very striking diseased appearance, without obvious derangement of anatomical position; there was a red grey change of the calamus scriptorius, so that the nib of this was quite involved, and from the nib upwards and outwards for half an inch there ran this change.

The lining membrane of this ventricle and its choroid plexus were of a deeper colour than usual.

Müller gives a case in full detail, and considers that the real seat of the disease is in that part of the encephalon, which regulates the articulation of words and the voluntary part of swallowing.

A large number of cases are on record that are wanting in completeness from the absence of post-mortem examinations. In some the orbicularis oris was paralysed, but not the buccinator; and the nerves affected show every degree of abnormality.

Closely connected with these cases are those in which one or more of these nerves are affected. I was lately asked to see a man who had had hemiplegia for some time. The day before I first saw him he had another fit, and in addition to the loss of motor power in the limbs he lost the power of swallowing. To a great extent he was able to speak, and he moved his tongue with ease. He died of exhaustion consequent on the impossibility of deglutition.

In a case recorded by Mr. Henry Taylor, a man after a fall complained of great pain over the left side of the occiput, and in a few days' time found he had entirely lost the power of deglutition. He complained

of numbness of the right arm and leg, but had perfect motion. The pupil of the right eye was dilated, but sensible to light. He expressed himself as feeling quite well, only very hungry. He made the most determined efforts to swallow, but the fluid always returned by the nose. He sank from exhaustion. On removing the brain at the necropsy, that organ was found perfectly healthy, with the exception of the left vertebral artery, which was entirely filled up with an embolic clot.

Lichtenstein records three cases in which what he calls *laloplegia* or *glosso-plegia* was the most prominent symptom, two of them recovering before long completely, and one dying. He believes that it may occur as a temporary affection from mental emotion, or as a permanent one from some exudation at the site of the nervous centres which preside over speech. This he locates at the upper part of the medulla oblongata, near the origin of the vagi, which are often involved to some extent at some period of the affection.

A somewhat anomalous case bears upon this subject:—

A boy, five years old, bright and intelligent in mind, but having always been extremely delicate, and liable to faint away on the least exertion, is the subject of the following symptoms:—He has never been able to swallow anything solid since his birth. He swallows liquids well. The palate is equal, and is moved on inspiration. He protrudes the tongue well and straight. He cannot pronounce D or K. There is a little

diminution of the motor power of the right facial muscles. No others are paralysed. Septum nasi is pushed over towards the left side, and nearly fills up the left nostril. Cries, laughs, and coughs much as other children.

The intimate relation between the nuclei of the nerves generally implicated in glosso-laryngeal paralysis explains anatomically the most usual association of symptoms.

Lockhart Clarke has demonstrated a close anatomical connection between the nuclei of the hypoglossal, vagus, spinal accessory, facial, and trigeminal nerves. He says there is a column of cells forming the nuclei of these nerves which supplies all the parts used in speaking. The whole of this group of cells being diseased, we get loss of deglutition, loss of voice, loss of speech in great measure, especially of labials, loss of motor power in the lips, tongue, and palate.

Part of the group only being affected, we get partial symptoms, loss of deglutition associated with the power of protruding the tongue, loss of phonation with comparative preservation of the motor power of the lips, &c.

The affection is distinguished from paralysis of the soft palate succeeding to simple or diphtheritic angina by the lips and tongue not being involved in the latter; from double facial paralysis by the electric sensibility being lost in this affection, and not in glosso-laryngeal paralysis; from general paralysis of the insane in that in this latter disease there is no special difficulty in articulating certain letters, and the movement of the tongue is quite free.

What then is the special lesion? It is in the nerve-roots, an absolute or partial disappearance of the nerve-tubes, the neurilemma being left, though in some cases stained and altered. In Dr. Wilks' case the roots of the hypoglossal and spinal accessory nerves had lost their natural white opaque appearance, and had become little thin gelatinous threads. The particular lesion of the nerves in this form, called by Jaccoud '*atrophie spontanée*,' consists not in a purely fatty degeneration, but in a fibrous fatty degeneration. In his plates he portrays a very considerable growth of fibrous tissue; the whole lesion being made up of steatosis and proliferation of connective tissue. In one of Trousseau's cases there was seen in the midst of the elements of the neurilemma a fatty substance, irregularly scattered in granules. The fibres of the connective tissue were markedly developed, and were mixed up with a good many elastic fibres. This form of degeneration may be due to inflammatory action; that is, it may be the latest development or sequence of slow inflammation, or it may be a purely passive process. When it does not depend on inflammation, it probably owes its origin to grey degeneration, or true sclerosis, of the group of cells forming the nuclei of these associated nerves. I say probably, because the absolute lesion has yet to be demonstrated microscopically, although in Dr. Wilks' case the medulla oblongata had a diseased appearance even to the naked eye. This is the more probable from the connection of this disease with progressive muscular atrophy. In several of the cases recorded the two conditions

were associated. In the former the paralysis is the first symptom, and muscular atrophy when it is met with is only towards the close of the disease; in the latter the muscular atrophy is the first symptom, and the paralysis seems generally to be in direct proportion to the loss of muscular tissue.

Duchenne considers that these two diseases are only associated accidentally. Still it is difficult, when they coexist in the same individual, to believe in a difference in the nature of the lesion. The atrophy of the motor roots may exist in each disease; in each there may be sclerosis of some portion of the nervous centres, of the medulla oblongata in the one, of the anterior columns and the grey matter of the cord in the other. The probable explanation of the connection is that the atrophy of the muscles depends on some lesion of the sympathetic that may or may not be present in glosso-laryngeal paralysis.

This is all that is known of the pathological anatomy of this disease, and the key-note of its simplification is in Lockhart Clarke's demonstration of the associated nuclei of these several nerves.

But still the question is not invariably so simple, since Voisin has recorded a case of glosso-labio-laryngeal paralysis without sclerosis of the medulla oblongata; and Jaccoud in his latest work says, '*Il n'est donc pas possible de faire de cette paralysie une entité morbide véritable, puisqu'elle n'est uniforme ni dans ses symptômes ni dans ses lésions: il y a là simplement deux modalités topographiques de la paralysie des nerfs bulbaires; au surplus, cette paralysie*

sous ou sus-bulbaire n'est pas toujours isolée ; on l'a vue coïncider avec la paralysie des nerfs oculomoteurs, avec l'atrophie musculaire progressive, enfin, avec une paralysie plus ou moins marquée des membres thoraciques.'

Facial Paralysis.—It is one of the inconveniences of speaking of diseases in this special way, that we have in talking of facial paralysis to include in it a great variety of morbid conditions.

In order that you may understand pretty easily the abnormalities to which the facial nerve is subject, it will be well to remind you briefly of its origin and its course. Here we again owe our thanks to Stilling and Lockhart Clarke for the discovery of the relations of the nucleus of the facial with the nuclei of other nerves. The hypoglossal, the vagus, the spinal accessory in part at least, the facial, and the fifth, all have nuclei closely associated with each other in the lower part of the medulla oblongata. Besides these, close to the olivary bodies, the sixth nerve arises from the same nucleus as the facial.

The nucleus of this nerve, therefore, is on the floor of the fourth ventricle, but some of its fibres are derived from the pons, even if a few do not come from an origin as high as the corpus striatum, or at any rate as high as the crus cerebri. In some of the lower animals, however, and to some extent even in man, some of the fibres of the facial nerve do not spring from the facial nucleus, but pass through the raphe of the medulla oblongata to the columns of the spine,

whilst those fibres, in man the most numerous, which spring from the nucleus, send radiating fibres across the raphe to the nucleus of the other side, so that in the medulla itself there is a double decussation. This decussation is somewhat higher than that of the motor fibres to the limbs.

The facial and auditory nerves appear together on each side of the posterior margin of the pons, opposite to its junction with the middle peduncles of the cerebellum. The facial is nearer the median line, and in its course with the auditory lies in a groove on the upper surface of that nerve. It runs outwards with the auditory to the internal auditory meatus, and at the bottom of the meatus enters the aqueduct of Fallopius, and follows the winding of that canal to the surface of the skull. At the bend, the nerve presents a reddish gangliform enlargement, which marks the junction of several nerves. These nerves are a large superficial petrosal from the Vidian nerve, a filament from the small superficial petrosal derived from the tympanic nerve, and the external superficial petrosal from the sympathetic accompanying the middle meningeal artery. Near the exit from the aqueduct of Fallopius it is joined by a few filaments from the auricular branch of the pneumogastric. In this osseous canal the chorda tympani is given off from the facial, supplying the membrana tympani, &c., and ultimately is inclined forwards to the gustatory nerve, ending in the submaxillary ganglion. Opposite the pyramid the facial nerve is arched downwards behind the tympanum to the stylomastoid foramen, by which it leaves the skull.

Outside the skull the facial nerve gives off branches in the following order : (1) The posterior auricular ; (2) the digastric and stylo-hyoid arising together ; (3) the temporo-facial, splitting up into temporal, malar, and infra-orbital branches ; and (4) the cervico-facial, passing through the parotid gland towards the angle of the lower jaw, and terminating in buccal, supra-maxillary, and infra-maxillary branches.

The lesions of the facial nerve are often divided into lesions inside and those outside the stylo-mastoid foramen. But the symptoms vary very much according to the position of the lesion in or near the nucleus, and in each part of its course.

Jaccoud's division is more in accordance with clinical observation. He would divide the causes of the paralysis of this nerve into cerebral and peripheral, and the peripheral are separated into basilar, interstitial, and superficial lesions.

Let me remind you that, whilst in facial palsy, from lesion of the portio dura, the orbicularis palpebrarum is almost invariably paralysed, in the great majority of cases of cerebral hemiplegia of the face, the orbicularis palpebrarum is not materially affected ; the act of winking and of voluntary closure of the eyes continues on the paralysed as on the sound side, with the single exception that the voluntary closure is usually weaker on the palsied side. These symptoms indeed, as Dr. Sanders remarks, form one of the best diagnostic marks between centric and peripheral paralysis of the face : the hanging cheek, with wide-open, staring, unwinking eye, denotes lesion of the portio dura ; the flaccid face,

with the natural position and motions of the eyelids, is a sign of cerebral lesion, and indicates a more serious disease.

Central lesions, confined to the nuclei of the nerve, are extremely rare. Hæmorrhage into that portion of the medulla oblongata would be very rapidly fatal. Still, judging by the analogy of glosso-laryngeal paralysis, a lesion that implicated these nuclei may exist. If the nucleus be affected it is almost certain that the sixth nerve, springing as it does from the same nucleus, will be affected also, and the patient will have converging strabismus.

Complete paralysis of the nerve has been known to coexist with hemiplegia of the same side. The explanation is difficult, and Dr. Brown-Séquard has suggested that the facial paralysis in these cases was due to reflex irritation.

In a few cases there is a cross paralysis seen, the facial nerve being paralysed on one side, the limbs on the other. This is invariably due to disease of the pons, and is easily intelligible anatomically. The wonder is that, with hæmorrhage into the pons, it is not more frequent. That it is not so is simply because a lesion to produce these symptoms must be in a very limited portion of the pons. In Dr. Hughlings Jackson's words : ' Since the nerve-fibres for the limbs cross below the pyramids, those of the left arm and leg will pass in the right side of the pons, on their way to the higher parts of the motor tracts. But the facial nerve of the right side runs through the lower part of the right side of the pons to its nucleus on the floor of the

fourth ventricle. So that a clot which damages the right facial nerve damages also the motor fibres which have come over from the left arm and leg.'

The morbid conditions that may influence the function of the nerve in the pons or the medulla oblongata are hæmorrhages, inflammations, softening, sclerosis, and tumours.

Disease of the motor tract above the pons varolii causes a facial paralysis very limited in extent and very slight in degree ; and the facial paralysis is, under these circumstances, always on the same side as the paralysis of the arm and leg.

Disease in the crus cerebri will cause hemiplegia and facial paralysis on the side opposite to the lesion, and if the disease extends to the nucleus of the third nerve in the crus, there will be paralysis of the third nerve on the same side as the lesion, and therefore on the opposite side to the hemiplegia and facial paralysis.

Lesion above this spot will implicate the facial nerve very slightly, and the facial paralysis may be therefore rapidly and almost completely recovered from. In disease, indeed, above the crus only a small part of the face, notably the levator anguli oris, levator labii superioris, and the zygomatici, will be paralysed, and in many cases not wholly so ; always, however, on the same side as the limbs, that is, on the side opposite to the lesion, showing not only that a few fibres of the facial exist above the crus, but that the fibres that are found higher up decussate eventually.

We may easily conceive that this very partial affec-

tion of the facial in lesion of parts above the crus cerebri is owing to the very limited number of fibres of the facial to be found above this point. A more elaborate explanation has been given, however, by Dr. Sanders, with the idea of showing why the orbicularis palpebrarum escapes, or at least is so very slightly affected, in ordinary hemiplegia. He says: 'In examining this question it is necessary to remember that one reason why the action of the orbicularis palpebrarum is not arrested in cerebral hemiplegia is, because ordinary winking is a reflex and not a voluntary act. When the motor influence of the brain is withdrawn from a part, the rule is that the voluntary movements cease, but the reflex actions persist. Winking continues therefore in cerebral hemiplegia of the face after the mobility of the features has ceased, for the same reason that the respiratory movements of the intercostals and diaphragm are preserved in the cerebral hemiplegia, which deprives the limbs of voluntary power. On the other hand, both voluntary and involuntary closing of the eyelids is prevented, and the orbicularis palpebrarum and other muscles are completely paralysed, in lesion of the portio dura, because the efferent conduction of all motor influence, reflex and voluntary alike, is then interrupted. In the same way spinal hemiplegia is distinguished from cerebral by the muscles of the thorax being paralysed in conjunction with those of the limbs, because in the spinal disease the tracts for reflex action are injured simultaneously with the conduction of volition. This explanation, which accounts for part of the phenomena, may be extended, however, beyond the simple reflex

action, as follows :—The facial motor or *portio dura* of the seventh pair, considered as a musculo-motor nerve, contains within its common trunk fibres serving different purposes, and provided, therefore, with different capacities of action. These fibres may, for our present inquiry, be divided into—1, voluntary motor fibres, by which the voluntary movements of the features are performed, and by which, especially, labial and buccal speech and mastication are accomplished ; 2, emotional fibres, by which the features express the passions more or less involuntarily ; 3, reflex motor fibres, which are involuntary, for the act of winking, and for the movements of the nostrils in respiration. According to the principles of physiology, these different sets of fibres derive their peculiar functions, not from any special properties of the nervous fibres themselves, but solely from the nature of their origin or central connections in the brain or the medulla oblongata. Thus, although anatomy cannot yet trace individual fibres to their primary source, there is good reason to believe that the voluntary motor fibres originate from a cerebral centre of conscious volition ; those for expression must be connected with the cerebral organs of the emotions ; while those for reflex action arise, more or less independently of the cerebral hemispheres, from the medulla oblongata, in connection (*inter alia*) with the sensory branches of the fifth pair, with which they are associated in the act of winking. The symptoms therefore will vary with the special seat of the central disease.’

I doubt, however, whether the phenomena demand so ingenious an explanation.

The lesions then in the brain itself affecting any portion of the portio dura directly must be in the medulla oblongata, the pons, the crura cerebri, the optic thalami, or the corpora striata. But any lesion of the cerebral hemispheres that causes either pressure or irritation of the corpora striata may indirectly induce slight paralysis of some fibres of the portio dura.

The nerve has, however, a short free course from the lower part of the pons, where it leaves the nervous centre, to its entrance into the petrous portion of the temporal bone. During this course it may be injured. Dr. Ogle has seen a case where the nerve was paralysed by being stretched by an aneurism of the anterior cerebellar artery. It may be injured by accident, such as fracture of the skull, by being pressed upon by meningeal hæmorrhage, or by tumour at the base of the brain, or by being imbedded in lymph from meningitis. In the Oxford Museum there is a specimen of a facial nerve as hard as cartilage. The disease was probably due to very gradual inflammation, secondary to ulceration in the internal ear. At the stylo-mastoid foramen the nerve came to an end, so that all communication with the face was cut off. The facial paralysis, however, would have been caused by the sclerosis of the nerve, even had not the peripheral destruction occurred. But injury or disease of the nerve in this short passage within the cranium is rare.

It is not, however, very unusual to meet with paralysis of the portio dura from disease in its course through the petrous portion of the temporal bone. The lesions are most commonly osteitis or caries of the pars petrosa,

otitis of the middle or of the internal ear, hæmorrhages in the aqueduct of Fallopius, and fracture of the temporal bone. In the majority of cases the portio dura will only be affected secondarily, the inflammation attacking the auditory filaments first and travelling backwards to affect the portio dura ; the occurrence of deafness affording aid to the diagnosis.

In other cases, especially where suppuration commences in the middle ear, we often find the membrana tympani destroyed, and discharge taking place from the external meatus. Not only do two filaments pass from the portio dura into the middle ear, but the whole relation of this portion of the auditory apparatus with this nerve is so close that disease of the middle chamber would seem to be particularly likely to cause facial paralysis. Still, as a matter of clinical observation, disease of this part is frequently met with without any facial paralysis. Deafness, followed later by facial paralysis, would suggest disease of the middle or internal ear, spreading afterwards to the portio dura. Deafness and facial paralysis occurring simultaneously, and unaccompanied by other paralysis, should suggest lesion of the two nerves as they enter together the pars petrosa ; and such a lesion would probably be either hæmorrhage or fracture of bone.

Anatomically, we should expect that lesion attacking the nerve during its progress through the bone would be evidenced by some paralysis of the chorda tympani, as this branch is only given off a very short distance above the stylo-mastoid foramen. It passes through the short canal behind the tympanum,

enters the tympanum, crosses it to the membrana tympani and the handle of the malleus, and emerges at the opening at the inner side of the Glasserian fissure; then lies beneath the external pterygoid muscle, and is inclined obliquely forwards to the gustatory nerve, a branch of the fifth, ending in the submaxillary ganglion. Observation seems rather to contra-indicate that the sense of taste is impaired in cases where the facial is seriously injured above the point of going off the chorda tympani. But, of course, we are almost wholly in the dark as to what part the chorda tympani takes in intensifying the action of the gustatory branch of the fifth. At any rate, it does not give us any definite aid in determining the seat of lesion of the facial nerve.

Jaccoud speaks of lesion to the nerve in its transit through the bone as evidenced by paralysis of the branches given off by the facial in the aqueduct of Fallopius; these phenomena being deviation of the tongue and difficulty in turning the point upwards, from paralysis of the branches to the digastric and stylo-glossus, and of the lingual branch; diminution of taste, from paralysis of the lingual branch and of the chorda tympani, the erector nerves of the papillæ; the exaltation of hearing, especially for the lower and highest notes of the scale, from paralysis of the small superficial petrosal, which supplies the internal muscle of the malleus; diminution of the salivary secretion and dryness of the mouth, from paralysis of the chorda tympani running to the submaxillary ganglion, and of the small petrosal going to the parotid; and lastly, the

alteration in shape of the velum of the palate, from paralysis of the large superficial petrosal nerve. This deformity consists in a deviation of the uvula, which sometimes is turned wholly towards the healthy side; sometimes its point deviates towards the paralysed side, whilst its base is turned towards the other. At the same time, the velum palati is enfeebled and flabby on the diseased side.

I can only say that English observers have been very much alive to these points, and have found taste frequently unaffected; and many of them believe, with Dr. Todd, that the affection of the palate is of very rare occurrence, and then only as a coincidence; but Dr. Sanders describes a case in which this condition of the palate coexisted with facial paralysis, and, as he thinks, depended upon lesion of this nerve.

Lastly, we come to affections of the nerve after it has emerged from the stylo-mastoid foramen. This is what is generally understood by true facial paralysis; and the abnormal condition is generally very complete, involving the filaments that supply the orbicularis palpebrarum quite as much as those which supply the muscles round the mouth. The electric contractility of the nerve is in complete abeyance.

The kinds of lesion are manifold :—

1. Injuries to the nerve by various accidents or by surgical operation. Before Sir Charles Bell's beautiful elaboration of the various nerves of the face, section of the facial had been attempted for the cure of tic. The nerve may easily be injured by operation, even the most careful, for the removal of tumour in the neigh-

bourhood of the styloid process. There have also been instances in which the nerve at its exit has been injured by the forceps of the accoucheur in a tedious birth, though this accident is much more likely to affect some branch of the temporo-facial termination of the nerve.

2. Swelling of the parotid gland, or tumours in its immediate neighbourhood. Part of the difficulty of opening the mouth in ordinary mumps is due to the pressure exercised by the gland on a portion of the nerve.

3. Syphilis. In this case the paralysis is generally very localised from the pressure upward on the nerve in spots where it is very near bone.

4. Exhaustion. Dr. Todd mentions this as an occasional cause. It ought, perhaps, to be placed among the central lesions; as also may the next, to which, again, Dr. Todd has drawn the attention of the profession.

5. Paludal poison: the motor power of a motor nerve being affected by marsh-miasm, and paralysis induced, just as neuralgia is caused by the action of the same poison on a sensory nerve. Still, neither exhaustion nor paludal poison can be supposed to act only on the peripheral portion of a nerve; they must act by causing anæmia of the nucleus.

6. Rheumatism or gout, as distinguished from the impress of cold; that is, paralysis of the facial nerve due to the rheumatic diathesis. Here, again, it is perhaps hardly fair to speak of this as being a cause of peripheral paralysis. Dr. Todd's case 16 is an example of this.

7. Cold. The most frequent of all causes; so much so, that peripheral facial paralysis has been called a disease only of the northern climates. Sometimes the paralytic condition comes on quite suddenly, after the patient has been exposed to great cold, especially to the influence of a cold draught impinging immediately on the trunk of the nerve as it emerges from the stylo-mastoid foramen. In this case the nerve trunk is simply much congested. At other times the paralytic symptoms come on more gradually, and the interior of the nerve in that case is the seat of an exudation of serum or of lymph; in a word, it is an instance of neuritis.

If the paralysis persists, we either get a condition of sclerosis of the nerve—the result of low subacute inflammation—or more or less atrophy of the nerve, due partly to the pressure of the exudation, partly to disuse of the nerve itself.

It is in such cases, where the nerve has undergone persistent degenerative changes, that we may meet with contraction of the paralysed muscles, the spasmodic tic of Marshall Hall.

A few cases have been recorded of double facial paralysis. Very seldom are both the muscles affected simultaneously. If they are, we may expect the lesion to be an aneurism of the basilar artery, or central hæmorrhage of the lower part of the pons, or an accident causing fracture of each pars petrosa. The double paralysis is sometimes seen when the lesion is in the temporal bone, being then attended with deafness. Cases have been met with depending on

rheumatism or on syphilis, and in each case affecting only the peripheral portion of the nerve. Dr. Warburton Begbie has seen one example of double facial palsy occurring in a phthisical patient, who subsequently died of what appeared to be strumous meningitis; but no autopsy was permitted.

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LECTURE X.

PROGRESSIVE MUSCULAR ATROPHY.

EVER since this disease has been recognised there have been diverse views as to its pathology ; some observers considering the degeneration of muscles the main, and sometimes even the sole lesion, others finding it in some portion of the nervous system. Since Lockhart Clarke's observations, however, most authorities unite in considering the nervous system to be the seat of primary lesion, but there is no complete agreement as to the portion of the nervous system affected.

Still, taking into consideration the comparative agreement of a majority of recorded cases, and the probability of imperfect observation of some other, it may be said that the most usual, I cannot say the universal, lesion is atrophy of the anterior grey cornua.

The lesions that have been observed in the spinal cord are mainly these : softening of the centre of the grey matter in the cervical and dorsal regions ; atrophy of the grey matter with amyloid deposits and granular bodies ; this atrophy has been found to extend to the anterior cornua, to the posterior, to both at one time, to the posterior commissure, to the central part itself,

which may have been already encroached upon by enlargement of the central canal. Lockhart Clarke has found changes in the grey matter. Round or at the sides of bloodvessels spots were seen differing from proper grey matter, and composed of a substance which had a delicate transparent and very finely granular aspect. A considerable deposit of corpora amylacea existed round the central canal. These appearances, however, also existed in parts of the cord, from which the nervous influence was derived to perfectly healthy muscles. Lastly, grey degeneration of the posterior columns with amyloid bodies, under which circumstances the symptoms of locomotor ataxy and progressive muscular atrophy are found combined. In many cases, lesions of the anterior nerve-roots have been found, and this lesion consists in a diminution of volume caused by the disappearance of a certain number of the nerve-tubes. This alteration may be more or less seen in various parts of the cord, but especially in the cervical region. It is important to remember that these roots contain, besides the motor filaments, some filaments of the sympathetic. Before I say anything about the sympathetic, let me point out to you that the above-named lesions are very diverse, and seem to deny to the anterior cornua any specialty as the seat of lesion.

Besides which the fact observed by Dumenil and the more recent observations of Bamberger seem to refute it; in these cases the cord was examined microscopically by Recklinghausen, and was found completely healthy. In one of the cases, the anterior roots

were alone diseased ; in the other, in which the atrophy of the muscles was less advanced, these roots were quite normal, but in the most altered muscles he found degeneration of some of the nerve-filaments. The result of these two post-mortem examinations was such that Bamberger concluded from them that in progressive muscular atrophy the process begins by degeneration of the muscles, and that the alteration of the peripheral nerves and of the anterior roots are secondary lesions. That the disease of nerve-root might be secondary, depending on the disease of the muscles it supplies, is only analogous to what is seen in the body. The optic nerve will become atrophied when the eye is lost, and in some cases, at least when a limb is lost, the nerve atrophies also.

The examples of lesion of the sympathetic in this disease amounted to six up to the date of the publication in 1873 of 'Jaccoud's Volumes on the Pathology of Internal Organs.' The first in date was that of Schneevogt ; the two following were observed by Jaccoud in 1864 ; two belonged to Dumenil, and the last was observed by Swarzenski in Frerich's clinique. In Schneevogt's case, the fatty degeneration of the sympathetic occupied the cervical and dorsal cords ; in Jaccoud's cases, there was a fibro-fatty degeneration of the sympathetic cord in the neck of the superior cervical ganglion, and (in one of them) of the branches that unite this ganglion and the spinal cord. In these three cases, atrophy of the anterior root existed in various degrees, but the spinal cord was healthy ; in Dumenil's observation he found a selero-fatty atrophy

of the cervico-dorsal sympathetic, and a granular fatty alteration of the lumbar sympathetic, coincident with atrophy of the roots, and with many lesions of the cord, and even of the intervertebral ganglia. Swarzenski states that in his case the two superior ganglia of the sympathetic were much flattened and of a livid colour, but no microscopical examination was made. The explanation of this lesion in its relation to progressive muscular atrophy is thus given by Jaccoud: 'Independently of the motor and sensitive filaments, the muscles receive by the nerves that emanate from the cord sympathetic or trophic filaments, which maintain and rule their nutrition; complete inertia of a nerve, therefore, has necessarily as its effects the loss of motion, the loss of sensation, and insufficient nutrition (atrophy). In the trunks of the nerve the three filaments are closely grouped together; they cannot be attacked in an isolated way, so that the complete lesion of such a nerve has always the triple result I have indicated; but if there is a point at which these various elements can be separately altered, the effect will be simple instead of being complex; this will be, according to the case, either a loss of motion alone, or a loss of sensation alone, or, lastly, an alteration of nutrition, an atrophy alone.'

Now this region of isolation really exists; for the motor filaments, it is the anterior spinal system and the anterior roots as far as the intervertebral ganglia; for the sensory filaments, it is the posterior spinal system and the corresponding roots; for the trophic filaments, it is the whole tract they pass through before their fusion

with the peripheral nerve-trunk—that is to say, the grey substance of the cord, wherever they take their origin, the anterior and posterior roots by which they leave the spinal axis, the anastomosing branches which connect the cord with the sympathetic, the sympathetic cord itself, and the branches which spring from it. In all these points the lesions of nerves may be isolated, and in this case it has for its sole consequence insufficiency of nutrition : if, further, this lesion is extensive or progressive, the greater part of the animal muscular system is attacked with atrophy without primary or contemporaneous paralysis either of motion or of sensation. This is precisely the clinical characteristic of the disease, the anatomical and pathological condition of which is a lesion of the sympathetic or trophic system.

The state of the muscles is one either of fatty or granular degeneration, and of these the latter may be only a first stage of the former. We observe at first a disappearance of the transverse and longitudinal striæ, whilst at the same time the contents of the sarcolemma present as it were a granular segmentation ; they are replaced by opaque granulations of a nitrogenous nature, which are dissolved for the most part in acetic acid, and are not modified either by ether or by alcohol. The muscular bundles then undergo a considerable diminution in volume. Later on these granulations undergo the fatty transformation, and some fat is also deposited in the muscular interstices, constituting what is called interstitial fatty degeneration, as opposed to parenchymatous degeneration. Even if we look at

these two lesions as distinct and isolated, or as necessarily succeeding each other, we have just the same reason for recognising in muscular atrophy two dissimilar alterations, the granular and the fatty, this latter presenting two forms generally combined, the parenchymatous and the interstitial form.

Finally, atrophy, the disappearance of the contractile element, may still be produced by another mechanism, that is, by the proliferation of the connective elements which enter into the composition of the muscle. When this substitution is complete the muscular fibres are transformed into true fibrous cords. This lesion may coincide with fatty degeneration in the same subject, but it may also exist alone. This muscular sclerosis appears to correspond with the third form of atrophy, described by Professor Robin as a whitish transformation. It is of great importance to recognise this special lesion; and Jaccoud believes that it enables us to conceive the possibility of permanent muscular retractions in progressive atrophy, and furnishes a satisfactory interpretation of this symptom. Dr. Bazire, in a note to Trousseau's article on this disease, says that in his opinion it is still an open question whether this atrophy is an idiopathic affection of the muscles themselves, or whether it is dependent on structural alterations of the spinal cord. He states that Dr. Meryon, who holds the former opinion, has supported it by the results of two carefully made microscopical examinations of the cord in undoubted instances of the disease, and even cites a case in which the bones seemed to participate in the inadequate nourish-

ment of the muscles. In 105 cases collected by Dr. Roberts, in his essay on wasting palsy, the cord was examined in 13 and found altered in 4 only. But Dr. Roberts, in a later article in 'Reynolds's System of Medicine,' gives a very different relative proportion of cases in which the cord was affected. He says, 'The condition of the spinal cord and of the spinal nerves has been examined in thirty-five cases, of which thirty-four have been tabulated by Bergmann.' The results of the investigation have not been by any means uniform. In sixteen cases the cord and the nerves were pronounced healthy, and in six of these the parts were examined microscopically. In six cases the cord itself was found healthy, but there was marked atrophy of the anterior roots of a certain number of spinal nerves. In one case both the spinal cord and nerves were healthy, but there existed disease of the medulla oblongata. In six cases the cord was found diseased, when examined microscopically, though it appeared sound, or nearly so, to the naked eye. Lastly, in several cases the cord appeared to the unaided senses palpably softened and disorganized. Dr. Bazire thinks that the total absence of all visceral disorders in this complaint is a fact not easily reconcilable with an affection of the sympathetic, and he says, 'It is a remarkable circumstance that pale unstriated muscular fibres escape in progressive muscular atrophy, and that the heart, the walls of which consist of striated fibres, is not affected. But this is only in accordance with the capriciousness of the line of attack, which is a striking characteristic in this disease.'

When we consider the difficulty of examining every portion of the spinal cord in any one case, and the frequency with which lesions have been found in this organ, I believe the seat of lesion must be placed in the cord itself. Still, it need not be at first such as would cause paralysis, either of motion or sensation. Mr. Lockhart Clarke, speaking on this point, says, 'There may be very obscure structural changes in the grey substance of the cord, or perhaps only in the ganglia or the posterior roots of the nerves, that may affect the nutrition of the parts to which they are subservient, without interfering with the functions either of sensation or motion; and in cases where the lesions occur in small isolated spots, the limitation of disease to particular muscles, or even to particular fasciculi of any one muscle, could be explained, I think, by the particular nerve-fibrils within the grey substance.' This abnormality of the trophic nerves, or of the trophic centres, will cause atrophy of some kind or other of the muscles; this atrophy, leading to disease, causes, or may cause, atrophy of nerves or nerve-roots, in accordance also with the clinical observation that the paralysis follows the loss of muscular substance, and is always in direct ratio with such loss. Further loss of power will also be induced by extension of original lesion in the cord to those portions of the grey matter that rule motion, or to the anterior columns, as the tract by which the orders of will are transmitted downwards. It is only by further extension of the same lesion to the posterior columns that we meet with the

combination of the symptoms of locomotor ataxy with progressive muscular atrophy.

M. Gombault has recorded a case of this disease, accompanied by glosso-labio-laryngeal paralysis. In this case the nerve-cells of the hypoglossal nucleus in the medulla oblongata were found to have undergone considerable pigmentary degeneration, to have lost their angular appearance and some of their processes; whilst in the spinal cord both the antero-lateral column and the grey substance were considerably altered by disease.

Locomotor Ataxy.—It may be well, as an aid in understanding the pathological anatomy of this disease, to mention briefly its most important symptoms:—

1. Certain affections of the eyes; diplopia, strabismus, partial loss of sight, complete amaurosis, and contraction of pupils. All these ocular symptoms may be absent or intermittent; or they may occur and vanish, and not recur again. A man lately in the Bristol Royal Infirmary with locomotor ataxy had complete amaurosis for ten days, and entire recovery from it.

2. Neuralgic pains of very severe character, especially in the limbs about to be affected. These pains are often afterwards followed by a corresponding numbness.

3. Want of coördination in the muscles of the limbs, with all the varied phenomena consequent on this condition.

4. Sometimes, but very rarely, affection of the special senses other than sight, especially taste and smell.

5. Towards the termination of the disease certain paralysees affecting the sphincters, the muscles of the limbs, particularly of the legs.

It is scarcely necessary to refer you to my Lectures of the Winter Session, in order to remind you of the differential points between this disease and chorea on the one hand, and disease of the cerebellum on the other. Suffice it to say, that there are most important points of difference between these three morbid conditions.

The morbid appearances are, lesion of the posterior columns, of the posterior roots of the spinal nerves, and probably of the posterior cornua. Dr. Axenfeld's account of the lesion (quoted by Trousseau) is as follows:—‘ In the white matter of the posterior columns, which has now become yellowish or grey, are seen scattered nerve-tubes, pale, shrunk, or varicose, sometimes reduced to their neurilemma only, or filled with granular contents, a few still retaining their axis cylinders. On the other hand, the connective transparent substance, the blastema in which these tubes are imbedded, has become fibrillated, and presents, amidst a large quantity of amorphous granules, a smaller quantity of elongated nuclei, and a smaller one still of cells (perhaps the nuclei, or at least most of them, belong to the nerve-sheaths). Corpora amylacea also are met with in variable quantity, distinguishable by their usual reaction with tincture of iodine. Lastly, the blood-vessels are considerably developed, and their thickened walls, composed of several layers, are incrustated with a deposit of fatty granules. In the posterior cornua of the grey matter

the same alterations are found, but less markedly. The reddish tint of this part is due to the injection of its capillary network, and occasionally its tint is darker, blackish, owing to the presence of numerous granules of pigment. The nerve-tubes in these cornua are sometimes destroyed, and the nerve-cells altered in shape, although in general both the tubes and cells are normal. The changes noticed in the posterior roots are the same as those of the corresponding columns; and they are the same again in the diseased portions of the bulb, the pons, and the optic nerves.'

Lockhart Clarke's account is still more circumstantial: 'The morbid anatomy of locomotor ataxy consists chiefly of a certain grey degeneration and disintegration of the posterior columns of the spinal cord, of the posterior roots of the spinal nerves, of the posterior grey substance or cornua, and sometimes of the cerebral nerves. A variable number, and frequently, in the latter stages of the disease, nearly all the fibres of the posterior columns and posterior roots fall into a state of granular degeneration, and ultimately disappear. Usually the posterior columns retain their normal size and shape in consequence of hypertrophy of connective tissue, which replaces the lost fibres. In this tissue, at wide but variable intervals, lie imbedded the remaining nerve-fibres, with the débris of their neighbours in different stages of disintegration. In some places they are severed into short portions, or into rows of globular masses, formed out of their medullary sheaths or white substance, which has been stripped from their axis cylinders. In other places

they have fallen into smaller fragments and granules, which either lie aggregated in the line of the original fibres, or are scattered at irregular distances. Corpora amylacea are usually abundant, and oil-globules of different sizes are frequently interspersed among them, and collected into groups of variable shape and size around the blood-vessels of the part. I am inclined to believe, from my own investigations, that in the course of the disease the posterior cornua of grey substance are more or less affected; and it appears to me to be a question whether they are not the first parts, or at least among the first parts, that are morbidly changed. I have also shown that in some cases the deeper central parts of the grey substance are more or less injured by areas of disintegration. These latter lesions, however, are not essential to the production of locomotor ataxy, the peculiar symptoms of which depend solely on lesions of the posterior columns, of the posterior nerve-roots, and probably of the posterior cornua. The cases in which they occur may be considered as mixed cases, partaking of the nature of locomotor ataxy and common spinal paralysis.'

The lesion is grey degeneration, or a more intense form of sclerosis. (See plate 7.) Now, these lesions seldom affect the whole length of the cord, but they may do so. Generally only a limited portion of the cord is affected, and that most usually the dorso-lumbar, and very rarely the cervical portions.

In a case under my own care, however, the whole length of the cord was affected, but the cervical portion not so intensely as the lumbar and lower dorsal. The

patient had symptoms of locomotor ataxy for some years, and eventually a certain amount of paralysis. There was great diminution of the multipolar cells of the grey matter. The white matter, especially of the posterior columns, was almost wholly gone, its place being taken by many amyloid bodies, granular matter, and a considerable growth of the very finest connective tissue.

There are a few other points that demand remark. Thus the spinal membranes in some cases show marks of inflammation. In one of Trousseau's cases, the dura mater spinalis was very vascular in its whole length, and of a dark red hue; it was also very markedly thickened in its upper portions, and somewhat œdematous. There was no trace of old exudations. The spinal pia mater was also abnormally injected, more especially over the lower third of the cord and along its posterior columns.

In these points, in fact, it strongly adhered to the posterior column, and like them had a yellow tint. It could not be separated from the cord without tearing away some of the nerve-substance.

Again, the atrophy of the nerve-roots bears no accurate relation to the lesion of the posterior columns. The columns may be in a very distinct state of grey degeneration, and yet the nerve-roots connected with them be healthy.

Again, the course of the lesion seems to be different according as it attacks the cord or the brain. In the cord the central portions seem to be the first attacked, and the disease radiates towards the circumference; in the brain we find just the reverse; the periphery of

the nerves suffers first, then the body of the nerve, and then the centre from which it springs, as from the optic nerve to the corpora geniculata.

Again, when sight is lost or gravely impaired, there is a gradual whitening of the optic disc, without any diminution in the size of the retinal arteries and veins.

The lesion of the optic nerve is a progressive grey induration, and differs from optic neuritis.

The ophthalmoscopic characters correspond with what is understood as progressive atrophy of the papilla. It is a very interesting observation of Charcot's that the great majority of women who are admitted into the wards of the Salpêtrière suffering from amaurosis present sooner or later after their admission more or less marked symptoms of locomotor ataxy.

The form of sclerosis found in this disease, and called grey degeneration, has been termed by Rindfleisch non-inflammatory. The spinal meningitis which accompanies this sclerosis of the posterior columns, and which is usually limited to those parts, would seem to render it very probable that the disease has an inflammatory origin. Besides which, one part of the cord may be in a state of this grey degeneration, and another in a condition of the so-called inflammatory sclerosis.

It is not very likely that the origin of the lesion will be inflammatory in one part of the cord and non-inflammatory in another part.

Again, Charcot and Vulpian have pointed out that in general locomotor ataxy a degeneration of a special portion of the posterior columns is the characteristic and essential lesion. It is known that in cases of this

kind, there are to be observed, in addition to the sclerosis of the fillets of Goel, which is almost always present, two slender grey bundles. These, viewed on the surface of the cord, appear to occupy the posterior lateral fissures, and the most internal sensory roots appear to emerge from them. Degeneration of these bundles is then the special characteristic of the disease under consideration.

Another point in the pathological anatomy of this disease is with reference to general paralysis of the insane. Quite apart from the special mental difficulties in cases of the latter disease, of which exaggerated optimism is perhaps the most frequent, the muscular trembling, especially of the lips and tongue, the hesitation and mumbling of speech, with a peculiar unsteady gait quite different from the loss of coördination in locomotor ataxy, will suffice to separate the two conditions. But I cannot remind you too often that nature constantly shows us disease in a complex form. You have already seen that locomotor ataxy may, toward the end of its course, come to be mixed up with spinal paralysis, from extension of this lesion into the thickness of the grey matter of the cord. This mingling of two pathological conditions may be seen also in general paralysis of the insane; and Dr. Patrick Nichol has reported several cases in which the two states were coexistent. At the autopsy of one of them were found lesions characteristic of the two conditions: 'The pia mater thickened and white over the whole brain, but most so over the anterior and middle lobes. Four ounces of fluid escaped during removal; the con-

volutions much wasted. A pale pinkish tinge in the grey matter ; puncta vasculosa numerous ; brain substance watery ; ventricles very large and filled with clear fluid ; the ganglia which project into the lateral ventricle have lost their fulness and roundness ; numerous small holes visible to the naked eye in the substance of the corpora striata. On removal of the spinal cord, and opening of the sheath of dura mater, the arachnoid was found to be much thickened, and of a milky-white appearance. At the cauda equina, and over the lumbar enlargement, there are several dilated vessels gorged with blood. Extending up the posterior columns there was a softish white substance passing irregularly from side to side, commencing over the lumbar enlargement, being thickest throughout the dorsal region, and becoming thinner again in the cervical. It seemed to be under or in the visceral layer of the arachnoid, and resembled a streak of thick arrowroot-milk in appearance. There seemed to be some dilatation of the vessels on each side of it, and especially in the neighbourhood of the origin of the posterior roots of the nerves. The ligamenta dentata were thickened, whitish, and opaque. Microscopically, the lesion, grey degeneration, was seen to involve the posterior columns, and to reach the grey commissure, and partly to involve it, and also most of the rootlets of the posterior roots.

Dr. Crichton Browne has made the remark that in all the cases of locomotor ataxy with mental derangement that have fallen under his observation, and in which there was optimism, the optimism has been invariably tinged with suspicion. It is probable that

general paralysis is not the only form of mental disease with which locomotor ataxy may be associated.

In connection with this subject a very interesting point is the existence of cases which recover, cases therefore in which we may presume no grey degeneration has taken place in the posterior columns or the posterior cornua.

What is the lesion here? Excluding simulation and hysteria, are the ataxic symptoms due to reflex action, the irritation being in some distant organ; or are the phenomena due to anæmia of the cord of a temporary nature; or are they dependent on an exhausted state of the nervous centre, irrespective of blood supply, and leaving no recognisable lesion behind; or are they due to syphilitic lesion that may be cured? The great improvement that occasionally takes place, even where the cord is disorganised, in certain symptoms of ataxic patients, as for instance in the sight, or in the power of retaining the urine after there has been incontinence of urine for some time, seems to point to some non-persistent condition of these centres as sometimes occurring. Trousseau gives a case of this disease in which the most careful microscopical examination of the cord failed to disclose any lesion. Dr. Wilks reports three cases in which improvement was so marked under treatment as to amount to recovery. Are not such cases examples of what we see in many diseases of the cord and of the brain, the latter especially, that the pathological anatomy of many maladies of the nervous system exists only during the life of the patient.

Infantile Paralysis.—Here also, as in other morbid conditions of the nervous system, the pathological anatomy is twofold. In one set of cases, that do not improve under treatment, lesions are found of a very definite nature. In others, that completely recover, the temporary lesion is believed to be spinal congestion. The reasons for this belief are: (1) that an imperfect and curable form of paralysis does occur from spinal congestion. (2) That the symptoms of paralysis from spinal congestion and infantile paralysis are very similar. In both the paralysis is partial, in both sensation is exaggerated rather than dulled in the paralysed parts, in neither are the sphincters affected, in both the paralysed muscles are limber, not rigid, in both recovery more or less complete is the rule, in both head symptoms are at any time exceptional phenomena.

Dr. Hillier has tabulated the lesions that may be found, or may be supposed to exist, in this paralysis of children:—

1. It may be caused by an organic change in the muscles, as in inflammation of the muscles, and possibly in progressive muscular atrophy. .

2. By an organic change in the conductors of motor impulse, as of the nerves supplying the muscles, or part of the cord, or of the brain; either of these may be affected by inflammation, hæmorrhage, or degeneration.

3. By an organic change in that part of the brain which is the organ of will; this and the preceding usually go together.

4. By a morbid change in the blood, as in typhoid, scarlatina, measles, diphtheria, or anæmia.

5. By a reflex influence transmitted from afferent to efferent nerves, and thence to muscles, or from some part of the brain or cord which is the seat of irritation. 4 and 5 are often combined.

This differs from cerebral paralysis in hemiplegia being rare, and in the absence of muscular rigidity, which often attends cerebral paralysis. The disease to which we give the name of infantile paralysis is wholly dependent on the spinal cord. There may be hyperæmia of the spinal meninges, though this is often absent. The fulness of vessels is seen much more in the grey matter than in the white; if only slight, it cannot be recognised except by careful measurement of the capillaries under the microscope; if intense, it is easily seen with the naked eye; when it affects the whole length of the cord, it does so with unequal intensity; the cervical and lumbar swellings are most likely to be found especially hyperæmic. If the congestion has been of long duration or frequently repeated, it will leave persistent capillary dilatation, and, as in other morbid conditions, may become the point of departure for grave alterations of tissue, myelitis, sclerosis, &c. It is with this more serious disease that we find easily recognisable lesions. Cerebral abnormalities are seldom met with; when they are, they are probably more by way of accidental complications. There is certainly no special lesion of the brain in these cases. Still Laborde found in two cases a small quantity of reddish serosity in the lateral ventricles, and Vulpian a slight softening with adhesion of the pia mater in the neighbourhood of the fissure of Sylvius, coinciding with hæmorrhage of the

corpus striatum and optic thalamus of the left hemisphere.

Medulla Oblongata.—There may be cellular atrophy in the pyramids and on the floor of the fourth ventricle at the level of the pons.

Spinal Cord.—The spinal meninges may be very vascular or slightly opaline, with a little thickening. The volume of the cord is variable. There is sometimes atrophy of the lumbar swelling, either when the cervical swelling is normal, or with a diminution in volume of the whole cord. Sometimes there is atrophy of the dorsal and lumbar regions in the antero-lateral columns on both sides, or on one; or there may be induration of the lumbar swelling, or an alteration of colour of the medullary tissue. The anterior roots are sometimes healthy, but they are generally atrophied, and unequally, according to the regions from which they spring. Sections of cord show to the naked eye in some cases the transparent gelatinous colour peculiar to sclerosis of the nervous centres, or a distinct brown tint of the two anterior cornua, with marked softening. Sometimes the atrophy of the grey matter of the anterior cornua or of the antero-lateral columns can be thus seen.

Under the microscope we find a dissemination of lesions in the various regions of the cord. These lesions are situated in the anterior cornua of the grey matter, sometimes in the posterior commissure, but very rarely in the posterior cornua. The main lesion is a striking diminution in the volume of the anterior cornua of the grey matter, especially transversely. The groups of large motor cells in the anterior cornua have disappeared,

especially the internal group. The motor cells which remain are altered. Their dimensions are reduced in every way ; the prolongations of the cells being gone, some cells have no nucleus, or one smaller than normal. The nucleolus is not always present, and its colour is often changed. Between the nucleus and the walls of the nerve corpuscles there is often a mass of ochrey pigment.

The neuroglia is often condensed, but it may be in just the opposite condition, or else the reticulum is crowded with nuclei, which are much more numerous than in the parts that have remained healthy.

The vessels may not be appreciably altered, or else the lymphatic sheath of the vessel may be the seat of proliferation of the nuclei, or of crystals of hæmatoidine and masses of granular bodies. On sections the lumen of the small arteries may be seen to be obstructed by many granular bodies. These granular bodies may be numerous when the cells are little altered, or, on the contrary, very few may be seen when the cells of the anterior cornua are gone. Spots of softening are sometimes found near the vessels ; the myeline may be broken up in a state of small globular masses. The consistence of the tissue is variable. The granular bodies of ordinary softening are seldom found. These spots are never surrounded by any thickened wall or limiting membrane, as is often seen round spots of inflammatory softening. Some observers have remarked spots of red softening due to real myelitis surrounded by sclerosed tissue.

Hæmorrhagic spots or pigment, the result of old hæmorrhage, are occasionally among the medullary

lesions of infantile paralysis. Sometimes in the posterior cornua there is a partial or complete disappearance of the cellular group that constitutes the vesicular columns of Lockhart Clarke, either in the cervical or in the dorsal and lumbar regions.

The posterior commissure is sometimes traversed by a dense fibroid tract. In the white matter there may be an atrophy of the antero-lateral bundles only, either partial or general, due to sclerosis; but sclerosis may occur here without any atrophy. Or atrophy may be caused by extreme diminution in the volume of the axis cylinders. The nerve-tubes which go from the anterior cornua, and traverse the white bundles to become the anterior roots of the spinal nerves, may be attacked by sclerosis; and many amyloid bodies are found about these tubes.

The granular bodies and the alterations of the vessels described as existing in the grey matter are here met with equally.

The anterior roots have been found healthy; or they may be reduced to the axis cylinders, the myeline having disappeared; or the thin nerve-tubes may be separated by spaces filled with connective tissue.

Other lesions are found in the body.

1. The bones. There may be considerable diminution in their length and volume. The femur may be only the size of the humerus in the same subject. Luxations are common. The extremities of the limbs are pretty often considerably deformed. The cartilages lining the joints may be rough and granular. The epiphyses are of small volume, owing to arrest of

development. The atrophy of the bones bears no necessary relation to the extent of the muscular paralysis.

2. The muscles are generally in a state of fatty atrophy, or even of simple atrophy; or they may be granular and fatty, or with proliferation of the nuclei of the myolemma. The atrophy may not be immediately apparent, because the aponeuroses may be distended by fat. The muscles, on the other hand, may be healthy. When muscular disease is met with, the most common seat is of the antero-extensors of the leg, seldom of the foot; the thigh is affected less often than the leg; when it is, the muscles of the anterior region are chiefly attacked. Sometimes the psoas and iliacus, the intercostals, and the muscles of the trunk may be implicated; very seldom the muscles of the neck. The temporal muscle on one side of the face has been affected, but this is rare. In the upper extremity the extensors of the arm are generally first attacked, especially the deltoid, but sometimes the pectorales, the triceps, and the muscles of the thenar and hypothenar eminences.

3. The vessels that run to the atrophied part are sometimes diminished in volume, and occasionally sclero-atheromatous.

4. The peripheral nerves show some rarefaction of the nerve-tubes and increase of the connective tissue, or atrophy of the fibres, with proliferation of nuclei in the midst of these fibres.

The weight of authority is now tolerably united on the lesions of the spinal cord in infantile paralysis.

Heine (1840) and Duchenne (1855) referred its origin to the spinal cord, though they had no patho-

logical evidence of disease in the latter. Bouchut, however, placed the anatomical seat of the lesion in the muscles, and the disease among the muscular paralyses. Cornil, in 1863, and with Laborde in the following year, gave the result of their anatomical investigations; their example was followed by Prevost and Vulpian in 1865, Olivier in 1869, Charcot and Joffroy in 1870, and Parrot in 1871. Later still Damaschino and Roger have recorded three cases with autopsies. They state that the changes in the muscles were of the ordinary character, consisting of fatty and fibroid degeneration of the fibrillæ. In the spinal cord they found the same lesions of the grey substance and the anterior and lateral columns differing in the three cases only in its seat; in the first case lesions existed in two places, the cervical and lumbar enlargements; in the second, chiefly in the latter position; and in the third case, in the same region, but on both sides. The microscope revealed changes in the vessels, thickened walls, proliferation of their nuclei, atrophy of the ganglion cells and their processes, as well as of the nerve-fibres. There was also thickening of the connective tissue, especially marked in the third case. These facts lead these observers to the conclusions that the lesion of the spinal cord is essential and primary, and that the pathological appearances found justify their use of the term spinal infantile paralysis. The lesion they consider to be a myelitis, especially of the anterior grey substance, of which the atrophy of the nerves and muscles is the consequence. Rinecker gives the autop-

sies of two cases. In the second of the two, the anterior and lateral columns, as well as the nerve-fibres and ganglia, were atrophied, and in both the peripheral nerves. Rosenthal, instead of looking upon the atrophy of the nerve-cells as the primary cause, considers the dilatation and thickening of the vessels which he has observed, and which are the signs of an active participation of the latter, as a pathological process, which results further in secondary growth and destruction of the grey substance.

Feeling convinced, as I do, that infantile paralysis is due to lesion of the spinal cord, it is only fair to state that cases are on record in which no lesions can be found. Dr. Carl Kètli gives the history of a child who died of small-pox six months after the onset of paralysis. Professor Schenthauer undertook the microscopical examination of the spinal cord, which he found to be perfectly natural. Dr. Elischer examined the paralytic muscles with the following results: They showed two forms of pathological change, namely, fatty degeneration and a change which came nearest to colloid degeneration. The sarcolemma and nerves were not altered. In the striated muscles, instead of the single normal cell-nucleus, there were seen three or four granular cell-nuclei, which seemed to be at the same time enlarged, and contained two, three, or even more nucleoli. The contractile material was diminished, so that it did not fill out the sheath, but drew away from it. This atrophy was so great that at the upper and under part of the spindle-shaped cell-nucleus of the sheath there was hardly to be found a breadth of

·002 millimetre of cross-striped contractile muscular substance. Kétli thinks that these appearances point to an idiopathic muscular lesion, to peripheral and not central changes, as being the cause of the paralysis.

This and similar cases, however, may be explained on the view that the structural lesions were not found because they had not yet been formed. The child died of small-pox after having only for six months been subject to the paralysis. The lesions would very likely have only been dilatations of vessels, even if the child had not decidedly improved before it was taken with small-pox, of which we have no record.

Paralysis Agitans.—Although I have somewhat recently lost a case of paralysis agitans of long standing, death occurring from cerebral hæmorrhage, I have never had an opportunity of post-mortem examination in this disease; you must trust, therefore, to the observations of others. In some cases, generally those before middle age, recovery has taken place; and although the symptoms are in most respects similar, the pathology must differ from the cases of paralysis agitans that Trousseau well calls ‘senile trembling.’

Jaccoud's remarks on the pathological anatomy are as follows: ‘In certain cases the autopsy has shown no important alteration of the nervous centres even where there has been microscopical examination; in other and more numerous cases there has been observed general or diffuse induration (sclerosis) of the medulla oblongata, the pons, the corpora quadrigemina, sometimes also

of the cervical region of the cord, especially of the lateral columns. Various alterations have been met with besides, such as lacunæ in the meso-cephale, softening of the crura cerebri, simple rarefaction of nerve-tubes in the cord. In a case in which the symptoms of paralysis agitans had been confined to a right upper limb, the left optic thalamus was the seat of a sarcoma the size of a large nut. The inconstancy of these lesions, in the double point of view of their existence and their nature, takes away from them all specific character. Paralysis agitans is certainly a disease of the meso-cephale; but it is a neurosis, that is to say, a disease without any constant and uniform anatomical characteristics. Practically, however, it is of importance to remember that sclerosis of the meso-cephale is the alteration most frequently connected with this neurosis.' Drs. Murchison and Cayley record a case of paralysis agitans in which the spinal cord was examined after preparation. The cortical and connective tissue layer of the cord appeared thickened, and presented an increased number of nuclei. Irregular tracts and patches of connective tissue, thickly nucleated, passed from the cortical layer into the substance of the cord, the reticulum of which was much thickened. These patches were most frequently met with near the exit of the posterior roots of the nerves, which themselves did not appear altered. The place of the central canal was an oval tract, crowded with cells of various shapes and sizes, the majority having the character of leucocytes, none presenting the character of the normal epithelium of the canal. This oval tract occupied not only the site of the canal itself, but also

that of the surrounding central substantia gelatinosa. The capillaries of the grey matter, and to a less extent of the white, were distended with blood, and here and there were small points of extravasation. Through the whole cord, chiefly in the grey matter, were small deposits composed of leucocytes or exudation cells. The first three changes were due to a chronic, the last two to an acute process.

Joffroy's account of the morbid changes in these cases is very similar. In all there was proliferation of the epithelium completely filling up the central canal, great proliferation of nuclei in the neighbourhood of the ependyma, pigmentation of the nerve-cells, especially of those of Clarke's columna vesiculosa, and a large quantity of amyloid corpuscles. In the third case there was a sclerotic patch of connective tissue in the neighbourhood of the pons, with dilated vessels. The conclusion to which the writer comes is that the seat of the lesion in paralysis agitans is to be looked for in the cord.

In Oppolzer's case, quoted by Trousseau, the post-mortem appearances were as follows:—There was pneumonia of the right lung, and it is interesting that in Trousseau's three cases death had occurred from pneumonia. This coincidence is, however, only what is so frequently found in the subjects of chronic cerebral disease. The cranial bones were very thin, and their inner surface was rough. The dura mater was thickened, and adherent, here and there, to the inner table of the cranial vault; the pia mater opaque, and infiltrated with serosity; there was also a pretty large quantity of serosity in the subarachnoid cellular

tissue. The cerebral convolutions were thinner, the sulci between them seemed deeper than usual, the cortical substance was of a pale brown colour, while the medullary was perfectly white and traversed by dilated vessels; the cerebral substance was moist and of good consistence. The ventricles contained several drachms of transparent serosity, and the ependyma, principally on a level with the posterior cornua, was granular. In the substance of the right optic thalamus there was an apoplectic cyst of the size of a small bean, the walls of which contained pigment. The pons Varolii and the medulla oblongata were very manifestly indurated. The spinal cord was firm, and the medullary substance of the lateral columns, principally in the lumbar region, presented opaque grey striæ. On making a microscopical examination, there was found in the substance of the pons and of the medulla oblongata an abnormal production of connective tissue, accounting for the induration of those parts. The opaque striæ in the lateral columns of the cord were due to the presence of connective tissue in process of development.

Handfield Jones says that Stofella relates the case of a man, aged seventy-nine, who had the affection five years before his death. At the autopsy he found traces of atrophy of the brain, with secondary dropsy of the ventricles and of the cerebral membranes, and an apoplectic cyst the size of a pea in the right optic thalamus; the pons Varolii and medulla oblongata were remarkably stiff; the arteries at the base calcareous, and the lateral columns of the cord, especially

in the lumbar region, traversed by greyish opaque streaks, which, as well as the indurations in the pons and the medulla, resulted from embryonal connective tissue.

Of two autopsies recorded by Cohn, there was marked cerebral atrophy in one, and in the other wasting of the cord opposite the second cervical vertebra. The ages of the patients were forty-nine and seventy-four.

In Skoda's case, the brain was tough, the pia mater œdematous, the walls of the ventricles, the fornix, pons Varolii, medulla oblongata, and cord were remarkably stiff; both optic nerves were flattened and stiff. In some opaque reddish spots in the brain, the nerve-tissue was destroyed by embryonal connective tissue, which occasioned also the induration of the pons and medulla. The muscles were very fatty, the neurilemma of the nerves of the upper limbs was thickened.

It is worthy of remark that the tremor precedes the paralysis in point of time. At first in very many cases there is no absolute loss of motor power. Indeed the muscular power may be considerable. It is only later on that paralysis supervenes. This is important in connection with the observation that before middle life some of these cases are curable, even when not dependent on mercurial poisoning. It is impossible that in such cases sclerosis of any part of the brain or cord can have existed. The symptoms in such cases must be due to some abnormality of circulation, which may disappear under suitable treatment. Some de-

range of the nutrition of the nerve-cells, either from some abnormality of the blood itself or of the vessels, or some condition of the nerve-cells preventing proper assimilation of nutritive material from the blood, must obtain in these cases; and, as we have seen in other diseases of the nervous system, lesions recognisable after death are only produced by one or more of these abnormalities being prolonged beyond a certain time, and the nutrition being thereby materially and persistently affected.

That paralysis may depend upon an abnormal condition of circulation in the nervous centres, and may be recovered from when this is removed, is seen in spinal paralysis depending on congestion of the spinal cord; and tremor is so closely connected with paralysis, and is so often followed by it, that it is not straining analogy too far to believe in a similar cause in varying degree inducing both conditions.

But in cases that have gone too far for cure we may be pretty sure we shall find sclerosis of the mesocephale, and generally of some portions of the spinal cord.

This will probably explain the reason for MM. Bourneville and Guerard drawing the distinctions they do between paralysis agitans and cerebro-spinal sclerosis. A patient may recover from paralysis agitans, or may die with it, and no lesion be found; but if life is prolonged, lesions will be gradually induced, and these generally in the form of partial and scattered indurations. These observers are, however, right in saying that nystagmus (oscillation of the eyeballs) seldom, if ever, occurs in paralysis agitans.

Lead Palsy.—In lead-poisoning we may meet with the well-known symptoms of colic, with an aggravated dyspepsia, with anæmia, with cerebral phenomena taking various forms, manifesting themselves either as delirium, convulsion, or coma; neuralgia followed by paralysis of nerves or of parts of nerves, and of those nerves that supply the extensor muscles sooner than those which supply the flexors, as in wrist-drop; and, as a sequence of this paralysis, considerable atrophy of the muscles themselves.

In certain cases, before paralysis occurs, we find partial or general tremor, that is, tremor of groups of muscles, or of one limb, and tremor of all the limbs and even of the whole body.

Sometimes also amaurosis is met with, depending upon optic neuritis, or on atrophy of disc.

The brain is pale and soft, the convolutions wasted, the sulci wide, with serous fluid distending them, and separating the pia mater and the brain. Sometimes there are patches of white softening in the brain. We meet also with œdema of the brain leading to capillary anæmia from pressure; though this result is doubtful. There may be some periarteritis. Lead has been found in the brain.

The ganglia of the sympathetic have been found in a state of sclerosis. The nerves themselves are sometimes seen surrounded by small particles of lead, compressed, and with their nutrition interfered with and more or less atrophied.

I believe the nerves of the limbs are sometimes in a state of neuritis. This condition has been seen in

the retina in lead-poisoning. Dr. E. Meyer has recorded the cases of two sisters employed at Brussels in bleaching lace with the aid of white lead. In the first case the girl was attacked by colic at the age of nineteen, and afterwards became hemiplegic on the right side. From this she perfectly recovered, but her sight gradually failed, and in the course of four months she became perfectly blind. When the author first saw her the optic discs were in a state of complete white atrophy. The second sister, when twenty years old, suddenly fell down unconscious, and was convulsed. On recovery her mind was confused and her speech embarrassed. Another attack occurred a few hours later, and was followed by deep sleep. On awaking she complained of severe headache, and of a constantly thickening mist before her sight. On the day following she described herself as being blind, but when Dr. Meyer visited her two days later she could see the glimmer of a lamp two yards distant. The field of vision, tested by means of two candles, was considerably but irregularly contracted. The pupils were largely dilated. The ophthalmoscope showed the optic discs much swollen, elevated, opaque, and of a reddish-grey colour. The retina near the disc was opaque; the veins were large, flexuous, and deeply coloured; the arteries fine. The conditions were those described by Von Gräfe as due to neuritis, from obstruction to the circulation by constriction in the sclerotic ring.

Kussmaul and Maier have recorded a case of a house-painter who, with many symptoms of chronic lead-poisoning, yet had no paralysis or brain symptoms.

The post-mortem appearances seen are, however, very instructive: 'General and great emaciation; marked rigor mortis of the muscles and heart nineteen hours after death; a certain amount of jaundice; great catarrh of the stomach, intestines, and ductus choledochus; fatty degeneration of the glands of the stomach: slight fatty change of the muscular walls of the stomach, especially at the pylorus; atrophy of the mucous membrane of the jejunum, ileum, and upper part of colon, in which both the stroma and glands shared; atrophy of the intestinal villi, the glands of Lieberkühn, the solitary glands, and Peyer's patches; marked development of the submucous tissue of the stomach, and even more so of that of the intestines, from proliferation of the areolar tissue, and thickening of the sheaths of the vessels; this was shown especially in the smaller arteries in the narrowing of their calibre, caused by the large amount of fat cells deposited in the distended network of this layer; fatty degeneration of the muscles of the intestines, especially in the small intestines; pigmental degeneration of the muscular fibres of the heart. The brain, especially in its cortical substance, showed slight periarteritis; there was proliferation and sclerosis of the connective tissue septa of the small ganglia of the sympathetic, especially the celiac and the cervical; these glands were hard, and the circulation in them affected, and their nerve-cell elements diminished. The author considered that the changes found in the absorbent apparatus account for the chronic dyspepsia, the anæmic colour, and the malnutrition of the patient.'

These lesions account for many of the ordinary phenomena.

Besides these lesions, in experimental researches upon dogs, in chronic lead-poisoning the bones contained the largest quantity of lead, next the liver and the kidneys.

Heubel attributes the rapid atrophy of the muscles in lead-poisoning to general interference with nutrition, which lead causes in a marked degree, and partly to the paralysis itself, which he attributes to the direct action of the poison on the intra-muscular ends of the nerves, and not on their central ends.

A complication not unfrequently met with in lead-poisoning, but only indirectly connected with it, is granular degeneration of the kidney. It is so common an accompaniment that some physicians have considered the cerebral phenomena to depend upon uræmia. Still it has been easy to disprove this, since these symptoms have occurred without the presence of albumen in the urine; and such cases have shown after death no granular degeneration. But the frequency of the occurrence of this renal disease is very remarkable, and has an important bearing on the true pathology of lead-poisoning.

I am bound to tell you that the presence of lead in the various organs is not sufficient to account in any direct manner for the phenomena. I have no hesitation in saying that the cerebral atrophy, the cerebral anæmia, or the somewhat rare cases of cerebral softening, cannot owe their origin to the presence of lead in the brain. There must be some other cause, some-

thing more directly affecting the nutrition, than such a lesion as this.

Other phenomena of lead-poisoning point in the same direction. It is well known that women who work in lead manifest a great tendency to abort ; and, what is far more remarkable, if a man, the subject of lead-poisoning, begets a child, such a fœtus is comparatively unlikely to arrive at perfection. In fact, the impregnated ovum passes away as an abortion, if either parent is the subject of lead-poisoning. This surely cannot be explained by the mere presence of lead in this or that organ. Neither can the atrophy of the gastric and intestinal glands be explained on any ground, except that of a deteriorated blood. Still less can the frequent complication of gout. A very large proportion of workers in lead die of kidney disease ; a very large proportion of those who thus die have been attacked with gout. Gout associated with lead-poisoning seems to have a special affinity for the kidneys ; whilst gout associated with alcoholic excess will generally attack the joints.

The lesions in the brain, therefore, the atrophy of the gastric and intestinal glands, the granular degeneration of the kidneys, the tendency to abortion, the frequent coexistence of gout, all point to one and the same idea—that the blood is first injured by the lead-poisoning, and that most of the lesions above mentioned are due to abnormalities of nutrition, owing to this alteration of the blood.

That which has seemed necessary theoretically for the reasonable explanation both of the lesions and of

the morbid phenomena has received important confirmation by the observations of M. Malassez. He states that M. Andral long ago pointed out that the blood of patients suffering from lead-poisoning was deficient in red corpuscles. M. Malassez has, however, taken the trouble in eleven instances to count the absolute number of red corpuscles, and he finds that whilst men in good health have on the average 4,500,000 and women 3,500,000 red corpuscles in a cubic millimetre of their blood, the number fell in nine men affected with symptoms of saturnine spanæmia to 3,700,000, to 3,200,000, and even in one case to 2,200,000; whilst in two women the numbers were 2,900,000 and 2,500,000 respectively. M. Malassez further states that, if the red corpuscles are less numerous, they are more voluminous than natural in lead-poisoning, in the proportion of nine to seven. Nevertheless he estimates that the increase of volume is not sufficient to compensate for the diminution in number. If the augmentation in the size of the corpuscles has some advantage in compensating to a certain extent for the diminution in number, it has probably the opposite effect of rendering the blood less fluid, since the corpuscles would necessarily pass more slowly in capillaries but little larger than themselves, and would likewise occupy time in adapting themselves to curves and angles. He sums up the results of his observations: that saturnine intoxication causes a certain number of alterations in the blood, of which the principal are diminution in the number of the corpuscles, augmentation of their size—such increase being, how-

ever, insufficient to compensate for their diminution in number—a great fixity of their morphological elements, and finally, very probably a diminution in the circulatory activity.

Consult, for progressive muscular atrophy—

- Jaccoud, 'Path. interne.'
 Trousseau, 'Clin. Lect.,' and note by Dr. Bazire.
 Roberts on 'Wasting Palsy.'
 Reynolds, 'Syst. of Med.' Dr. Roberts.
 Dr. Duchenne, 'Atlas of Photographs.'
 'Med. Chir. Trans.,' li. & lvi. Lockhart Clarke.
 'New Syd. Soc. Yearbook,' 1862, p. 103.
 'Med. Record,' i. 10. M. Gombault.
 Ibid., i. 54. Lockhart Clarke.
-

Consult, for locomotor ataxy—

- Trousseau, 'Clin. Lect.'
 Hammond, 'Dis. of Nervous System.'
 Jaccoud, 'Path. interne.'
 Reynolds, 'Syst. of Med.' Dr. Radcliffe.
 'St. George's Hosp. Rep.,' v. 1. Lockhart Clarke (and pamphlet).
 'Journal of Mental Science,' Jan. 1874. M. Charcot.
 'Guy's Hosp. Rep.,' xvii. Dr. Wilks.
 'West Riding Asylum Rep.,' i. Dr. Nichol.
 'Path. Soc. Trans.,' xxii. 14. Drs. Greenhow and Cayley.
 'Irish Hosp. Gazette,' March 15, 1874. Bouchut.
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Consult, for infantile paralysis—

- Petitfils (monograph).
 Reynolds, 'Syst. of Med.' Dr. Radcliffe.
 Jaccoud, 'Path. interne.'
 Hammond. Op. cit.
 West, 'Dis. of Infancy and Childhood.'
 Hillier, 'Dis. of Children.'
 'New Syd. Soc. Yearbook,' 1871-2, p. 96. Damaschino and Roger.
 'Med. Record,' i. 102. Vulpian.
 Ibid., i. 424. Kètli.

Consult, for paralysis agitans—

Trousseau, 'Clin. Lectures.'

Jaccoud, 'Path. interne.'

Handfield Jones, 'Functional Nervous Disorders.'

Reynolds, 'Syst. of Med.' Dr. Sanders.

Bourneville and Guerard, 'De la Sclérose.'

'Guy's Hosp. Rep.,' xvii. 191.

'Path. Soc. Trans.,' xxii. 24.

'New Syd. Soc. Yearbook,' 1871. 98. Joffroy.

Consult, for lead palsy—

Hammond, 'Dis. of Nervous System.'

Reynolds, 'Syst. of Med.' Dr. Sanders.

Romberg, 'Dis. of Nervous System.'

Jaccoud, 'Path. interne.'

'Lancet,' May 16, 1874. M. Malassez.

'New Syd. Soc. Yearbook,' 1871-2. Kussmaul.

LECTURE XI.

EPILEPSY.

IT was an old saying of Van Swieten : ‘*Dum viso epileptico territus homo corripitur eodem morbo, quis definire audebit quid tunc mutatum fuerit in corpore.*’

With all our better means of research, we must confess at the present moment that the pathology of epilepsy is still very uncertain, whilst the pathological anatomy is simply confusing. Let us try to reach light out of so much obscurity ; and, first, we will mention the lesions that have been found post mortem in epileptics.

For instance, all parts of the brain have been found to be the seat of lesions of the most varied kind : foreign bodies developed on the meninges, in the ventricles, in the cerebral substance ; increase of sub-arachnoid fluid, or distension of the ventricles by serum ; induration, softening, and general swelling of the cerebral mass ; general or partial hyperæmia, cysts, tubercles, cancers, exostoses, periosteal growths, thickening, or some change of the arachnoid or the pia mater ; abnormal thickness or abnormal thinness of the cranial bones ; excessive size of head, increase in the volume of the cranial cavity, deformities, or abnormality in the conformation of this cavity ; caries

of the cranial bones; pus between the bone and the dura mater; acute or chronic hydrocephalus; hydatids; ossification of the dura mater, tubercle of the dura mater, or pachymeningitis, *i.e.* inflammation of the dura mater; abscess in the cerebral tissue, spots or regions of hæmorrhage; various traumatic lesions; alterations of the pineal gland; inequality in weight and size of the cerebral hemispheres; various lesions connected with blood-vessels, aneurism, embolism, atheroma, increase in size of the capillaries in the medulla oblongata, fatty degeneration of some portion of the medulla oblongata; capillary dilatation in the pons and cerebellum, hæmorrhage of pons; anæmia of brain, either from disease of vessels, or dependent upon general anæmia, &c. &c. Almost every abnormality of the viscera in the other cavities of the body have been found in epileptics. They may show after death many lesions of heart and lungs, of the digestive and urinary system, and, perhaps, especially of the organs of generation. Every variety of blood may coexist with epilepsy; sanguineous plethora, gout, anæmia, spanæmia, after prolonged discharges, &c.; and although it may not be recognised post mortem, epilepsy may depend upon malaria, possibly from the hydræmia induced by malaria.

Dr. Crichton Browne concludes that hypertrophy and induration are the characteristic brain changes in epileptic insanity. 'The first effect,' he says, 'of the interrupted pressure which is applied to the brain in epilepsy, appears to be a genuine hypertrophy and augmentation in volume. But hypertrophy is generally

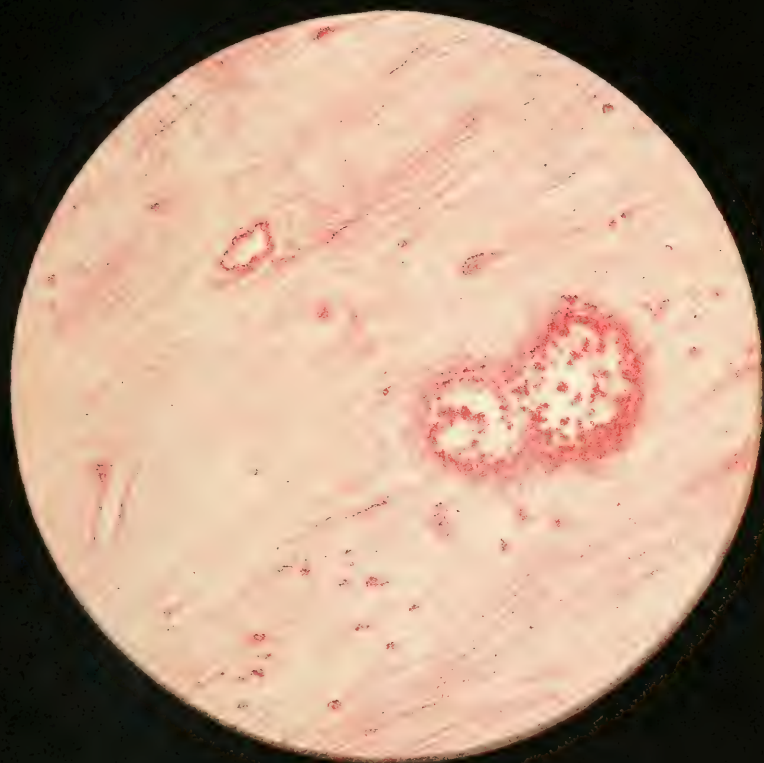
partial, and even when it affects whole organs it is manifested principally in certain textures; so this hypertrophy of brain in epilepsy is seen chiefly in the connective tissue. A kind of fibroid substitution slowly, but surely, goes on in those parts which are periodically subjected to congestion, and induration, as well as an augmentation in volume, ensues. The skull becomes thickened, and when it is removed the brain expands, as if relieved from compression, and feels unusually dense and hard when touched. The specific gravity both of its grey and white matter is greater than in any other class of lunatics, and the absolute weight of the brain is also decidedly higher. The convolutions are flattened, and the sulci are mere lines, and do not gape or contain fluid. The membranes show no signs of inflammatory disturbance. When the brain is cut into it is tough and firm, the grey matter being dark, and the medullary white and glistening. The ventricles are of small size. Around the pons Varolii and medulla oblongata, and especially on the floor of the fourth ventricle, redness and vascular dilatation are visible, and the vessels when measured are found considerably distended, owing both to increase in their sectional area and thickening of their walls. These are the usual appearances in the brains of persons who have laboured under epileptic insanity, but they are subject of course to numerous variations. Thus a spotty, blotchy, marbled appearance of the medullary substance may be seen when an attack or group of attacks has immediately preceded death, and some atrophy or wasting, with opacity of the arach-

noid, may be remarked when the disease has been long protracted, and has passed into epileptic stupor. This latter condition of the brain is referable to impaired nutrition, owing to the thickening of the vessels, or to gradual contraction of the hypertrophied fibrous tissue and puckering of the brain, if it may be so termed.'

And Dr. Crichton Browne also shows the true part played by pathological anatomy in this disease, when he says: 'With the clue that lies ready at hand in these coarse organic changes, we may proceed backwards to finer and less perceptible changes connected with earlier stages in epileptic degeneration; and from these, again, we may cautiously recede to those delicate and subtle changes which lie beyond our present means of exploration, and which are contemporaneous with what may be termed the functional epoch in the history of the disorder.'

Plate 16 shows a section of the medulla oblongata in a case of epileptic mania. In part of the field is seen a very dilated vessel. But the main lesion is a sacculated lacuna, caused by localized softening and absorption of the softened tissue.

In the report of the Somerset County Lunatic Asylum for 1864, Dr. Boyd gives the results of his observations upon 53 fatal cases of epilepsy. The shape and form of the skull presented an abnormality only in one case, in which it was thick behind, and the *diplœe* wanting. In nearly one-third there was a difference in the weight between the cerebral hemispheres of from $\frac{1}{2}$ to 6 ounces. Of the males 21 were epileptics only, and in them the



X 300

average weight of the brain was 50·3 ounces ; in 9 males who were epileptics and idiots the average weight of the brain was 46·6 ounces ; in 2 only of the latter it was below, but in 6 above the average. The average weight of the left cerebral hemisphere in the males was $\frac{3}{4}$ -ounce greater than the right hemisphere. Of the 23 females 20 were epileptics, and 3 epileptics and idiots ; in the 20 epileptics the average weight of the brain was 43·2 ounces, and the right cerebral hemisphere was slightly heavier than the left ; in the two idiots the brain was $\frac{1}{4}$ -ounce less than the average natural weight ; and in the third idiot, who died from cerebral apoplexy, which would add to the weight, the brain was $2\frac{3}{4}$ ounces heavier.

In 18 cases Messrs. Bouchut and Cazauvieilh found a true chronic inflammation of the cerebral tissue as the uniform lesion.

Wenzel's observations, accompanied by autopsies, amounted to twenty. He said that the pituitary body is invariably found diseased in epileptics, and the morbid condition almost invariably consists in an effusion of lymph which has become more or less indurated at the point of junction of the two lobes. Wenzel's opinion is that the slightest modifications of the pituitary body have the most serious consequences for the animal economy ; and that probably the diseased state of the pituitary body in epilepsy is the result of an inflammatory affection. But other observers have failed to verify Wenzel's observations in most cases. But after all, as Dr. Sieveking says, 'It is rather in its vital relations that the disease deserves to be studied

than in the dead-house'; and here again Dr. Thompson's observations of sphygmographic tracings show something of the pathology of the disease. He finds that the sphygmograph shows a lax condition of vessels, just the condition that is shown post mortem in the dilatation of the capillaries, especially in certain parts of the encephalon. It is in this line of work that it is possible to recognise the pathological conditions of epilepsy at what may be called the functional period of the disease.

Before we speak of the probable pathology of this disease, let us look at the successive phenomena of an epileptic fit. The following is the succession of cause and effect according to Brown-Séquard :

1. Excitation of certain parts of the excitomotory side of the nervous centre, leading to contraction of the blood-vessels of the brain proper and of the face, and spasm of some muscles of the eye and face.

2. Contraction of the blood-vessels of the brain proper, leading to loss of consciousness and accumulation of blood at the base of the encephalon.

3. Extension of the first excitation, partly due to accumulation of blood at the base of the encephalon, leading to tonic contraction of the laryngeal, cervical, and thoracic expiratory muscles.

(Called by Marshall Hall laryngismus and trache-lismus.)

4. Contraction of the laryngeal and thoracic expiratory muscles, leading to crying and stoppage of respiration.

5. Farther extension of the first excitation of the

nervous centre, leading to tonic contraction, extending to most of the muscles of the trunk and limbs.

6. Loss of consciousness and tonic contraction in the trunk and limbs, leading to falling.

7. Laryngismus, trachelismus, and a fixed state of the chest, leading to asphyxia, with obstruction to the return of venous blood from the head and spinal cavity.

8. Asphyxia and accumulation of black blood in encephalon and spinal cord, leading to clonic convulsions everywhere, contraction of the bowels, bladder, and uterus; erection, increase of many secretions, efforts at inspiration.

9. Exhaustion of nervous power generally, and of the reflex faculty specially, except for respiration, which gradually becomes normal, leading to a cessation of the convulsions, and to coma or heavy sleep, after which the patient complains of extreme fatigue and headache.

There is some objection to the view that clonic convulsion depends on the asphyxia and the accumulation of black blood in the encephalon and spinal cord. Brown-Séquard founds these views on some experiments in which clonic convulsions were set up on injecting venous blood into the system. It seems, however, doubtful whether these experiments bear out his conclusions. In Kussmaul and Tenner's experiments, the prevention of the entrance of red blood to the brain, when the veins were left perfectly free, induced violent convulsions. Again, in an animal bled to death, and in whom, therefore, no accumulation of

blood can have existed, the uterus expelled its contents precisely in the same manner as in an animal into whom venous blood had been injected. We would say, therefore, that clonic convulsions depend on the want of arterial blood in the spinal cord, caused partly by the further extension into the spinal cord of the first excitation (contraction of vessels), and partly by the asphyxia inducing a condition of venosity through the whole system.

If it is allowed that the loss of consciousness is occasioned by arterial spasm in the cerebral lobes, and that tonic rigidity and clonic convulsion are the consequence of an arterial spasm, similar in kind, if not in degree, in the medulla oblongata and spinal cord, it is difficult to believe that in the first case the function of the part is temporarily annihilated, whilst in the second it is intensely exalted. It is scarcely reasonable to suppose that the same condition which, when acting on the brain, causes the destruction of all cerebral functions, should, when acting on the spinal cord, cause an intense exaltation of the spinal function; nor can we believe that a fresh supply of arterial blood, which, acting on the brain, causes it to resume its functions, would, when acting on the spinal cord, almost in a similar ratio, lower and depress the functions of this new centre, as would be the case if the cessation of the convulsion were dependent on exhaustion of nervous power.

In illustration of the theory that clonic convulsions may be the consequence, not of excitation of a nervous centre by venous blood, but rather of a diminished

supply of arterial blood, I would mention an experiment performed by Vulpian, and recorded in his 'Leçons sur la Physiologie du Système Nerveux : ' ' J'ai vu chez un lapin la compression des artères carotides et vertébrales déterminer une suspension des fonctions encéphaliques ; mais, chose bien remarquable, la respiration spontanée continuait, le bulbe rachidien ayant échappé plus ou moins complètement à l'anémie encéphalique. Les mouvements spontanés et reflexes avaient entièrement disparu dans la face et les yeux ; le tronc de l'animal vivait encore en supportant une tête physiologiquement morte. Eh bien, au bout de deux ou trois minutes, les moyens de compression ayant été enlevés, il se produisit d'abord des mouvements convulsifs assez violents, qui ne durèrent que peu de temps ; puis toutes les manifestations de la vie, tous les mouvements volontaires et autres reparurent peu à peu dans le tête ; l'animal recommença à marcher et revint bientôt à son état normal.' Here the convulsions were the connecting link between apparent death or paralysis of the muscles of the head and face and their restoration to voluntary movement. Entire loss of arterial blood from the part was followed by loss of all movement ; the very commencement of restored arterial flow was followed by convulsion before the full circulation was restored ; and a full restoration of arterial circulation was the immediate precursor of normal life in the part.

Again, the explanation of these convulsive conditions seems peculiarly difficult, if we accept the theory that the accumulation of venous blood at the base of the brain is necessary for their production.

The following case I saw very frequently for two years :—

E. P., aged 61, a widow. Every day, and many times a day, for six months before I saw her, she suffered from a severe convulsive condition. She never loses consciousness in the least, and does not even feel confused; but has most violent clonic convulsions of the mouth, jaw, eyelids, and all the facial muscles. The head is shaken from side to side with intense rapidity; the arms and hands are in a similar state of clonic convulsion. The legs are not always affected; and when they are not she is able to stand through the whole of the attack. In all respects the convulsions of this case closely simulated epilepsy. She had no tonic spasm, no subsequent headache or drowsiness, no loss of memory or of any mental faculty, no paralysis. The absence of headache and sleepiness is a fair test of there having been no venous congestion of the brain. From a condition of ordinary healthy life she was wont to be taken with the intense clonic convulsion previously described, and the moment the convulsion ceased she was herself again.

The observation of all such cases seems to point to partial arterial spasm of the spinal cord as the proximate cause of the convulsion.

Several cases of a similar nature are collected from various sources in Dr. Reynolds' work on epilepsy.

It is true enough that some tonic spasm may exist and yet be very difficult of detection, and Trousseau would seem to look upon its non-existence as an impossibility. He says 'the tonic always precedes the

clonic stage; but the duration and violence of the latter are by no means proportionate to the duration and violence of the former. Thus, very violent clonic movements often succeed a slight tonic contraction, and reciprocally an excessively powerful tonic contraction may be succeeded by very moderate clonic movements. Thus the length of the first stage is sometimes so short, and the second stage comes on so quickly, lasting for a more or less prolonged period, that an observer who is not on his guard, or not very attentive, might think that the convulsions were clonic at the outset. From what I have said, this remarkable fact follows, that the rigidity seems to be an essential obligatory element of all convulsions. It is never absent, and can even be alone present, whether it constitutes the convulsion by itself, as in idiopathic contractions, or whether the convulsion is incomplete, as in eclampsia, where the clonic stage is absent, whereas clonic movements never, perhaps, come on from the first.'

This is a strong statement. In the case before mentioned the woman has more than once been talking to me in the out-patient room when the first symptoms of the attack have commenced in rapid twitching of the facial muscles, followed immediately by clonic convulsion of the limbs; and with the first twitching she has said, 'Now, sir, the fit is coming on.'

It is difficult to conceive how any person could speak with any laryngeal spasm sufficient to cause cerebral congestion; and if this venous blood theory be right, how is it that the tonic spasm and the clonic convulsion do not bear a direct ratio to each other?

A slight tonic stage should be followed by slight clonic convulsion, instead of the two stages bearing, as they often do, almost an inverse proportion to each other. How, too, can the period of rigidity exist by itself, unfollowed, as it sometimes is, by clonic convulsions?

I would rather repeat my belief that clonic convulsion is an evidence of partial spasm at the base of the brain and in the spinal cord; that it plays no part in the causation of the subsequent head-ache and stupor, except by sometimes adding the element of exhaustion when the convulsion has been very protracted; and that when the convulsion has followed the injection of venous blood, it has been from this venous blood inducing contraction in the arteries, as the blood in the exanthemata sometimes does.

Now with such a succession of phenomena, and such an explanation of them, there are three kinds of lesion for which we should search, both during the life of the patient and at the autopsy.

1. The cause of the abnormal irritability of the excitomotory system of nerves.

2. The excitants of the fit, which may be centric or eccentric.

3. Lesions, the results of the fit itself.

To take this last point first. A very large number of the lesions already mentioned come under this heading, especially those that have been found in the brains of insane epileptics. They may be divided for practical purposes into two classes, viz., lesions that depend on the mal-nutrition of the brain, consequent on constant repetition of arterial spasm and the anæmia

induced by it ; and lesions, the direct result of congestion and stasis, caused by the temporary asphyxia from the closure of the glottis and the tonic spasm of the muscles of respiration.

Under the first heading we meet with atrophy and white softening, with perhaps some increase of the subarachnoid fluid and some fulness of the ventricles ; under the second we meet with thickening of the cranial bones, adhesion or ossification of the dura mater ; pachymeningitis ; opacity of the arachnoid ; hæmorrhages from the vessels of the pia mater or in various parts of the brain, general or partial induration of the encephalon, absolute sclerosis depending on a very gradual form of cerebritis and persistent dilatation of vessels.

The difficult problem to solve in this relation is the reason why some epileptics escape these cerebral lesions for many years. It is quite certain that they may manifest no cerebral phenomena. Thus in a case lately seen by me in consultation, a lady of 27 years of age had had a severe fit almost every day of her life since she was three years old, and yet she was able to talk and reason as brilliantly as other people, her memory was good, and she showed no abnormality except irritability of temper. And this bad temper, although it may be due to commencing lesion in the brain, and culminate in a troublesome form of mania, yet in many cases it is only the natural result of the patient having to live so different a life from those around him, and being cut off from a life of useful occupation or amusement. The variation in point of

time at which such cases manifest cerebral lesion must depend on the greater resilience of arteries in some than in others, the vessel returning more easily and completely to its normal calibre and not becoming persistently dilated so early.

It is in direct accordance with the effects produced on the brain by the arterial spasm on the one hand and the temporary asphyxia on the other that we see the different results of the petit mal and the grand mal. In the former the fits may be very frequently repeated; and their constant repetition, meaning as it does frequently repeated arterial spasm of part of the brain and consequent anæmia, will produce a certain mental weakness much more quickly than the ordinary attacks of the grand mal.

These frequent slight fits interfere necessarily with the nutrition of the brain. But the asphyxia is so slightly marked and of so short a duration that the after effects on the brain, congestion, meningeal, or cerebral hæmorrhage, or inflammation, are absent. In le grand mal, however, all this is changed. The brain not only suffers from the arterial spasm and consequent innutrition, but from the effects of prolonged tonic spasm of the respiratory muscles; and instead of mere mental weakness we may meet with those cerebral phenomena that are found with hyperæmia of the cortical substance, or inflammation of some one of its layers, or induration. That these results are only induced gradually is because severe attacks of le grand mal are not usually repeated many times in the same day. The attacks, if not more or less periodic, are

generally separated from each other by a definite time—months, weeks, days, or at the least hours—whilst a patient may have several attacks of *le petit mal* in a single hour.

2. As to the excitants of the fit. This heading includes an infinite variety of lesions. It is difficult to say how far abnormalities of the blood should be included under this or under the first division. *Uræmia*, paludal poison, icterus, the presence in the blood of the virus of any of the exanthemata may induce the fit. The nutrition change of epilepsy may be a part of some general metamorphosis, such as that present in the several cachexiæ, as rheumatism, gout, syphilis, struma; it may be induced by some circumstances determining a relative excess of change in the medulla oblongata, or perhaps in the cortical substance of the cerebral hemispheres, during the general excess and perversion of organic change occurring at the periods of puberty, pregnancy, and dentition. The determining causes are here also probably a lesion of the blood itself.

It may be induced by anything that either increases the amount of carbonic acid in the blood, or, as I think, that diminishes the normal proportion of oxygen, such as various diseases of heart and lungs, alcoholism, pressure on the large arteries that feed the brain, &c. In children the mere presence of an abnormally heated blood in the brain will lead to epileptic phenomena. Undigested food in the stomach or bowels, worms, renal calculus, gallstones, various diseases of the liver, stomach, bowels, bladder, and very specially of the uterus and ovaries, may all act as exciting causes of epileptic phenomena.

The fit may be induced by conditions acting on the nervous centres directly, such as mechanical injuries, overwork, insolation, emotional disturbance, excessive venery, &c. It may be due to diseased action extending from contiguous portions of the nervous centres or their appendages. Thus we find it in meningitis, especially in its chronic forms, in aneurism or embolism of cerebral vessels, in clot in the brain, in any lesion affecting the various parts of the cortical substance. The diseased action may extend from various tumours, and perhaps also from exostoses or spiculæ of bone from the calvarium.

All these lesions may act as exciting causes of the phenomena, though several act also as predisposing; that is, the first and second divisions are much intermingled. Two factors at least are necessary for the production of an epileptic attack—the exciting cause, and an impressionable condition of the nervous substance, especially probably of the medulla oblongata. And this leads me to the real difficulty in the pathology of epilepsy, viz., what is the cause of the impressionable condition of the nervous system.

That which induces this arterial spasm, or rather that which causes the impressibility of the sympathetic nervous filaments, which rule the calibre of the arteries, must be the condition of the blood itself. It is from the blood that all the tissues derive the plasma for growth and change, and through the blood alone all remedies are brought to bear upon a disease. A morbid condition of this fluid will induce morbid mental phenomena entirely independent of any arterial

spasm or interference with its full supply. The observation of the many various forms of delirium teaches this most distinctly, that whilst some forms of delirium may depend upon a diminished supply of blood, a large number own as their cause the circulation of blood more or less diseased.

Twenty years ago, Mr. Harding, writing in the *Lancet*, made the following remarks: ‘The irregular muscular action of chorea has ever been regarded as connected with debility; the bloodless aspect in catalepsy bespeaks too plainly a poverty in life; the unsuccessful management of epilepsy by depletion led inquiring minds to investigate its pathology, and it is now looked upon as connected with a deficiency, not with an increase, in the amount of stimulus supplied by the blood.

‘The convulsions of young children occur in weakly not in strong subjects; and though the pulse may be quickened and congestion take place during the paroxysm sufficient to justify the application of the leech, it is essentially a disease of irritation as distinguished from inflammation.

‘If I may be allowed to pursue this reasoning further, let me mention the cramps which take place in elderly people, and where the diminished energy of the digestive power almost forbids the late and hearty meal. Again, the far more powerful and fearful cramp of cholera seems to commence and increase with exhaustion, when the blood has been drained of its watery particles by enormous discharges from the alimentary canal; and finally, the last act of the muscles commences when

exhaustion can proceed no more, assuming the cadaveric rigidity of structure when the vital spark has fled.'

As to the special lesion of the blood that induces this impressibility of the sympathetic or of the medulla oblongata, we are yet in ignorance; at any rate, we know of no lesion that invariably has this effect. But although the blood seems to be the special organ that induces this impressionable condition on the nervous centre, there are other facts to be considered. The whole question of hereditary influence is a case in point. But we shall probably ever remain in ignorance in this malady, as in gout, in rheumatism, in tubercle, in cancer, and even in syphilis, what this vulnerability means. A parent has a morbid constitution of some kind. He begets a child, and in so doing impresses upon the original germ, from which the child is developed, certain characteristics. Among these characteristics may be the likelihood of his offspring being gouty, but perhaps not till it has lived in the world for forty or fifty years. Or he impresses upon his offspring the peculiar abnormality by which a portion of a nerve centre or a special system of nerves may be morbidly liable to be set in action by almost any excitant, mental or physical. That the blood can have nothing to say to this question of hereditary influence directly is manifest by the anatomical fact that the blood of the child is of course not in existence at the period of conception. The impression is made on the cell, and it is only in process of development that it is, as it were, differentiated to the special part, just as any monstrosity may be transmitted from father to child. But, on the

other hand, it is quite as easy to believe that this hereditary vulnerability to a special disease is differentiated on the blood itself, and that the nervous system only suffers indirectly. And then again the very expression of an abnormality being differentiated on to a special organ is a convenient term by which we endeavour to indicate what later phenomena seem to shadow forth, but of the nature of which, or the mode of action of which, we are absolutely ignorant.

The blood, in strychnia-poisoning, has been found to be deficient in its power of assimilating oxygen. It is difficult to say whether this peculiarity in the physiological state of the blood may have been the cause of the lesions often found in the spinal cord in tetanus, of which I shall speak in another lecture. These lesions, it will be seen, are not constant, and are also found in other diseases in which there is no tonic spasm. It is at least probable that the tonic spasm, or rather the cause of the sympathetic filaments inducing tonic spasm, is this abnormality of blood. Now except in degree, and in the fact that different muscles are mainly attacked, the tonic spasm of tetanus differs in nature in no respect from tonic spasm that is one of the first phenomena of an epileptic fit.

Again, epileptiform convulsions are seen in people who are bleeding to death, in persons in whom the cerebral circulation is suddenly checked, in persons extremely debilitated by exhausting discharges. Epilepsy again has been seen in persons in whom there has been embolism of a middle cerebral artery, and in persons in whom there was aneurism of the same

artery, limiting almost equally the supply of blood to that arterial region. All clinical facts seem to me to point to the causation of epilepsy as depending either on a limitation of the supply of blood to some portion of the encephalon, or to an abnormality in the blood itself, and of all abnormalities the most important is the diminution of oxygen.

Any account of the pathology of epilepsy would at the present day be imperfect without some reference to Dr. Hughlings Jackson's views. Some of these views are necessarily speculative. He has made some observations on epileptic regions, and others as to the nature of the lesion. It is not easy to do full justice to the acumen of these views when stated in a tabulated form. Still it is necessary to make the attempt. Dr. Jackson believes:

‘ 1. That convulsion is a discharging lesion.

‘ 2. That since increase of function, even in disease, implies increased nutrition, we infer that the grey cells affected in convulsions store up force in large quantity, and reach a high degree of tension. Further, since they discharge on slight provocation, possibly even in periodical normal changes in the body, when by continuous nutrition a certain degree of tension is reached, we must suppose that they are in a state of highly unstable equilibrium.

‘ 3. That in epileptic discharges the convulsions usually begin in those parts of one side of the body which have the most voluntary uses. The order of frequency in which parts suffer illustrates the same law. Thus fits beginning in the hand are commonest. Next in frequency are those which begin in the face or tongue, and next are those which begin in the foot.

‘ 4. That many cases of epilepsy are due to some abnormality of the middle cerebral artery (aneurism, embolism, &c.).

‘ 5. That when epilepsy begins in the hand, and particularly if it frequently occurs in the same manner, and more especially if it sometimes remains localised in the hand and arms, we have every reason for diagnosing a discharging lesion of some part of the superior frontal convolutions of the opposite hemisphere—this has in some cases been found post mortem.

‘ 6. That epilepsy is the name for occasional, sudden, excessive, rapid, and local discharges of grey matter.

‘ 7. That, as any part of the grey matter of the cerebrum may become unstable, there will be all varieties of epilepsy, according to the exact position, or according to the extent of the grey matter altered, and there will be all degrees according to the degree of instability.

‘ 8. That we see externally that the stronger the spasm is, the wider spread it is ; the stronger the internal discharge, the further it will spread. There are two ways of spreading. The discharge will not only explode healthy lower centres, but will probably spread, as it were, laterally, to healthy associated centres in the brain. My speculation is that the latent spreading is by arteries and their vaso-motor nerves ; the spreading is in arterial regions.

‘ 9. That there are two ways in which nutrition may be imperfect, in quantity and in quality. I believe that nerve-tissue in discharging lesions is over-nourished in the former sense, and worse nourished in the latter. In order to make my meaning clearer, I will take chemical illustrations and use chemical nomenclature.

Two bodies may be of the same constitution, but yet of very different composition. For example, the constitution of acetic acid and of the chloracetic acid is the same, but they differ in composition, as in the latter hydrogen has been replaced by chlorine. The structure, however, is unaltered. I believe then that the highly unstable nervous matter of disease (as in a discharging lesion) differs in composition, but not in constitution, from the comparatively stable grey matter of health. The alteration in composition is of course such that the nervous substance found is more explosive. We must suppose that there is some order in this substitution-nutrition, and we must infer that it is in the direction of explosiveness or instability. The following is a speculation as to the kind of alteration of composition. One striking constituent of nervous matter is phosphorus. It belongs to the chemical class of triads, of which other members are nitrogen and arsenic. My speculation is that in the abnormal nutritive process producing unstable nervous matter the phosphorus ingredient is replaced by its chemical congener nitrogen. There is a substitution compound : the replacement probably occurs in different degrees, as it does in the three differing chloracetic acids. If nitrogen be substituted as supposed, we can easily understand that the substance produced would be more explosive. The supposed value of arsenic in certain nervous affections is significant, and it is another member of the group of triads. The nutrition is therefore assumed to be defective, not in quantity but in quality, in those functional alterations I call discharging lesions.' These are some of Dr. Hughlings Jackson's

views on epilepsy given partly in his own words. You see they bring us on another step in advance, first by proving as a matter of clinical observation that portions of the cortical regions of the hemispheres may be the originating seat of the epilepsy, either solely or primarily, and second, by giving us a valuable speculation, to the effect that the lesion may be an abnormal nutritive process, by which one ingredient of healthy nervous tissue has been replaced by another. We may say in passing that a substitution could scarcely occur, except from a faulty constitution of the blood itself.

It is of immense importance to know that some of Dr. Jackson's conclusions have been verified by the experiments of Professor Ferrier. He says not only that 'the anterior portions of the cerebral hemispheres are the chief centres of voluntary motion; but that the proximate causes of the different epilepsies are, as Dr. Hughlings Jackson supposes, discharging lesions of the different centres in the cerebral hemispheres. The affection may be limited artificially to one muscle or group of muscles, or may be made to involve all the muscles represented in the cerebral hemispheres, with foaming at the mouth, biting of the tongue, and loss of consciousness. When induced artificially in animals, the affection as a rule first invades the muscles most in voluntary use, in striking harmony with the clinical observation of Dr. Hughlings Jackson.'

And Professor Ferrier sums up his views on epilepsy thus:—

Many of the difficulties in the causation and definition of the epileptic state will disappear, if we

regard the phenomena of this disease or condition as proximately dependent on a local or general abnormal condition of the cerebral hemispheres, characterised by the tendency to recurrent sudden and explosive discharge of the motor centres, with or without functional perversion of the centres, which manifest themselves inwardly in consciousness. We shall thus be enabled to recognise in their true light, and place in their proper relations to the more generally recognised type of epileptic seizure, those forms of epilepsy which commence unilaterally in one or more groups of muscles, and which generally indicate some localised gross lesion of the cerebral hemispheres, while at the same time we shall afford a satisfactory explanation of the phenomena of idiopathic epilepsy, so-called, because it has not as yet been shown to be dependent on any constant discoverable lesion. In the local and unilateral attacks we have a successional analysis of the phenomena, which a so-called idiopathic attack presents in too complicated a form for successful diagnosis as to essential nature and causation. But in such cases also there are numerous facts which indicate that the seat of the motor and psychical disturbances of the epileptic attack is above the medulla oblongata, in centres which probably have an intimate relation with each other. And now that the motor signification of the grey matter of the hemispheres is clearly demonstrated, it is not necessary to assume that the medulla oblongata is the primary seat of the motor disturbances, while the psychical symptoms are only subordinate to changes induced in the circulation of the brain by a primary

affection of the medulla itself. When it is said that the proximate cause of the epileptic seizure is a condition of the instability of the cortical centres, nothing is implied as to the exact physical condition which may exist. It may be established as a hereditary tendency, and it is, we know, capable of being induced by numerous methods of centric or eccentric irritation, both in the indirect experiments of injury and disease, and as the result of direct experiments, such as blows on the head (Westphal), or injuries to nerve-trunks or the spinal cord (Brown-Séquard). To sum up then, in a few words, we may conclude:—

1. That an epileptic attack is a discharging lesion of some portion of the grey matter of the encephalon.

2. That a large proportion of lesions found in the brain post mortem are the result of the malnutrition of the brain by oft-repeated arterial spasm, or of congestion and further lesion of the brain, due to the spasm of respiratory muscles.

3. That certain lesions of brain may induce the impressibility of the grey matter, just as a clot rapidly breaking into healthy nervous tissue may sometimes produce convulsion.

4. That in some cases the cortical substance of the cerebral hemispheres, especially of the anterior lobes, in others the medulla oblongata or the spinal cord, may be the primary seat of the discharging lesion.

5. That the impressibility of these discharging centres is due to abnormalities of nutrition.

6. That these abnormalities of nutrition may be from variations in and especially deficiency of blood

supply, alteration in the character of the blood itself, particularly in its power of carrying oxygen, and perhaps sometimes in the substitution of an abnormal substance for one of the elementary constituents of the nervous tissue.

Chorea.—Trousseau says that, ‘as in the case of other neuroses, pathological anatomy teaches us scarcely anything as to the material alterations of the nervous centres in St. Vitus’s dance. If you consult various authors, you will find contradictory facts and opinions. One looks on inflammation or induration of the tubercula quadrigemina as the characteristic lesion of the disease; another regards as such induration or hypertrophy of the brain or of the spinal cord, or a more or less extensive softening of the cerebro-spinal centres; a third believes in calcareous concretions of the brain; a fourth in cysts of the pineal gland, or osteoids of the vertebral canal, and I know not what else. But does not this very diversity of the lesion found after death prove that there is no relation between them and the dynamic phenomena, even if it had not been ascertained that in most cases no appreciable anatomical change can be detected in the nerve-centres? For my own part, in the rare instances in which I have examined the bodies of individuals who had died of St. Vitus’s dance, after presenting the most violent symptoms of the disease, I never met with any lesion which seemed to me to be in accordance with the convulsive phenomena of chorea.’

Now, in considering the pathology of this disease, it is necessary briefly to review the circumstances under

which it arises. These are either moral, such as fright, conscious or unconscious imitation of another choraic patient, violent excitement such as has been seen in periods of great religious exaltation—as among the flagellants, among some of the subjects of the dancing mania of the fourteenth and fifteenth centuries, in the ranks of the Convulsionnaires of France, and in many religious revivals, especially among the Methodists, in Great Britain and in America—or physical, as chlorosis, or states closely related to it, or pregnancy, or rheumatism.

We shall see directly that rheumatism is connected with chorea in a special manner; that chorea follows it and seldom precedes it, and that in the rare cases in which it seems to precede it there is often evidence during life or after death that some valve or other of the heart has suffered, even though there may have been no manifestation of rheumatism in the joints or elsewhere.

There have been various statistics of post-mortem results in cases of chorea. Dr. John Ogle found, out of sixteen cases, congestion of brain or of spinal cord, or of both, in six; in one, actual softening of the spinal cord; in one, the spinal cord more dull and yellow than usual; in one, the central parts of the brain much softened; in ten, granulations on some part of the endocardium.

Among the records of the Pathological Society we find that fatal cases of chorea have presented the following lesions: Chronic disease of the cerebral dura mater, softening of the brain and spinal cord, tumours

in the centre of the spinal cord, softening of the fornix and of the surface of the third ventricle, softening of the crura cerebri with atrophy of the brain, softening of the central parts of the brain and of the corpora quadrigemina, the specific gravity of the corpus striatum and optic thalamus greater on one side than on the other, spinal meningitis, the arachnoid opaque on the surface of the hemispheres and serum in the ventricles, tubercle of the spinal cord, softening of the cord alone, the medulla oblongata pressed upon by an enlarged odontoid process, and, in chorea combined with paralysis, softening of the occipital cerebral convolutions with atrophy and degeneration of the spinal cord.

Skoda thinks the immediate cause of the disease is an exudation in the spinal cord or in the brain.

Hammond believes that chorea is either functional or organic; that in the former there are no post-mortem appearances, whilst in the latter form there may be—(1) intracranial congestion, softening, opacities, and adhesions; or (2) congestion or softening of the spinal cord with adhesions and opacities of membranes; or (3) embolism of the corpora striata with a beady mitral valve; or (4) hyperinosis of the blood.

I have been unable to trace any post-mortem appearance that would account for the phenomena in some cases, but in the following case there was evident lesion :

Girl, aged 17; has had rheumatism. Mitral disease evident during life, and the valve found beady after death. She seemed to die of exhaustion, as the choraic

jactitations were uncontrollable during the whole course of the disease until her death.

Some clots of blood were found lying outside the brain on the right side in the cavity of the arachnoid. The blood seemed to have come from a small orifice in the right lateral sinus. Many capillary embolisms in both corpora striata. The heart showed no beads of lymph on the ventricular surface of the mitral valve ; but on opening the left auricle the whole circle of the auricular attachment of the valve was surrounded by minute beadings.

The relations of chorea with rheumatism and heart disease in children have been minutely discussed by M. Roger. He says there is an interdependence between the three conditions. He admits a cardiac chorea, but says the heart disease and the chorea may start together from the same cause, as is the case sometimes with rheumatism and carditis. His cases establish that the heart disease often persists after the cure of the chorea ; and he further shows that chorea may be a cause of heart disease.

Dr. Murchison records the case of a girl, aged 14, who had for years suffered from chorea with mitral deficiency. While lying in bed she became suddenly unconscious, and had occasional muscular twitchings of the right limbs. The right pupil was contracted, the left dilated, both immovable. In addition to vegetations on the mitral valve were found embolic masses in the spleen and kidneys, and the left vertebral and left carotic arteries were much distended, hard, and completely blocked by a pale, firm, easily detached clot.

No embolisms of the minute vessels, such as have been described after death from chorea, were found.

We come then to the pathological theory, only partially as yet proved by post-mortem data, associated especially with the names of Kirkes, Tuckwell, Broadbent, and Hughlings Jackson, and spoken of on the Continent as the English theory of embolism.

This theory was shadowed forth by Dr. Kirkes without going much into particulars. His observations in the dead-house taught him that persons dying of chorea often showed on some portion of the endocardium, and particularly on the mitral valve, beady vegetations. Kirkes believed that these vegetations were washed away and carried by the blood to be deposited in some portion of the brain. Beyond this he did not go.

Dr. Jackson, however, goes further. Founding his views partly on clinical observation and partly on the experience of the dead-house, he suggests that these minute vegetations are indeed carried to the brain, as Kirkes supposed, and are there entangled in some portion generally of the middle cerebral artery, blocking up with emboli some one or more of its branches, and affecting therefore either the corpora striata or the convolutions in their immediate neighbourhood. He connects chorea with paralysis in that, whilst an embolus blocking up a large arterial branch will cause the latter, several minute emboli obstructing several small branches in the same situation will induce, not absolute loss of function, but impairment of function. His views are thus spoken of by Dr. Radcliffe: 'Taking

chorea of one side of the body, hemichorea, as the simplest form of chorea, and putting it side by side with hemiplegia, the result of embolism, good reason is found for believing that the disorder of movement and the palsy both point to the region of the corpus striatum as the seat of mischief. If this be the seat of mischief in hemiplegia, why not in hemichorea? The muscles most moved in hemichorea are those most palsied in hemiplegia. In hemichorea, as in hemiplegia, the arm, as a rule, is more affected than the leg. In right hemichorea, as in right hemiplegia, the speech is generally very much affected. Again, hemichorea is always more or less mixed up with, and sometimes ends in, hemiplegia; and, on the other hand, hemiplegia, from various causes, is not unfrequently attended with chorea, or movements of some kind or other. The fact that the face is involved in chorea shows that the seat of the disorder must be above the spinal cord. The facts which have been instanced point to the convolutions near the corpus striatum, rather than to any other part of the brain, as the part affected.'

Dr. Broadbent, theorising on perfectly independent data, would limit the portion of the nerve-centre affected to the sensory-motor ganglia; and he believes that the symptoms point to the seat of the mischief, not to its nature; and that besides embolism, hæmorrhage, softening, irritation, and other causes, may figure among the causes of chorea. He instances local innutrition, reflex action from peripheral irritation, and direct action upon the sensory-motor ganglia from shock.

His views that embolism is not the only lesion of the corpora striata in chorea are supported by a case reported by Dr. Foot at the Pathological Society of Dublin. He exhibited the viscera of a man, aged 68, affected with unilateral chorea, who had died of capillary bronchitis. On the surface, and towards the anterior end of the corpus striatum of the side opposite to that which had been affected, were two shallow depressions, underneath which were two deliberately encysted depôts of white diffuent brain substance, showing granular corpuscles with molecules and fatty débris. Running transversely across the deepest part of the interior of the optic thalamus of the same side was a deeply discoloured track, orange-red at the end approaching the lateral ventricle, plum-red at the opposite end where it approached the quadrigeminal bodies. Both basal and cortical arteries were almost without exception atheromatous. The spinal cord presented no anomaly; the cerebellum was soft and greasy; the kidneys were small, granular, and cystic; their cortex was reduced in thickness, their arteries were atheromatous. The left ventricle of the heart was one inch thick at apex and midpoint, seven lines at base; the larger curtain of the mitral valve was atheromatous, in fact, showing mitral valvulitis from the increased strain of an enlarged ventricle. The aorta presented all the various degrees of the atheromatous process.

There can be no doubt of two facts in the pathological anatomy of chorea: one, that embolism of some branches of the middle cerebral artery is a frequent lesion in this disease; and the other, that it is not the only lesion.

Dr. West states very forcibly the theoretical objections to the embolic theory.

1. 'The occasional occurrence of chorea from mere imitation, so that we have sometimes been compelled to change the position of patients in the Children's Hospital, from observing the involuntary mimicry by one child of the movements of another.

2. The extreme rarity of a sudden attack of chorea, the great slowness with which it almost invariably comes on.

3. The very small number of instances in which chorea continues limited to one side, and the comparatively short time within which hemichorea almost always becomes bilateral.

4. The almost invariable recovery of complete power over all the limbs in cases of chorea, and this even in instances where the paralytic character of the symptoms has most predominated ; so that I have only two or three times in my life met with cases in which permanent loss of power over a limb could be reasonably referred to antecedent chorea.

5. The fact that, as a general rule, and one with very few exceptions, the second attack of chorea is slighter than the first, and the third than the second ; a result wholly unintelligible, if organic mischief were the ordinary cause of the attack.'

I should myself be inclined to agree with Dr. Broadbent that embolism, though perhaps the chief, is far from being the only lesion ; and with Dr. Hughlings Jackson, that not the corpora striata only, but also the region of brain supplied by the middle cerebral artery

may be the seat of lesion. And without entering upon any controversy as to what kinds of lesion are capable of inducing the phenomena, it is sufficiently comprehensive to state that any lesion that interferes with the nutrition of this region of the brain may cause choraic jactitation, if only the interference with nutrition is not of sufficient extent to cause paralysis. It is only in this way we can account for the causation of chorea by shock and mental excitement of any kind; the direct mental influence producing arterial spasm, not only partial in degree, but specially limited in area. It is only on the ground of interference with nutrition that we can explain again the instances of chorea without embolism occurring in patients the subjects of chlorosis or of pregnancy. Indeed, chorea occurring during, and in consequence of, pregnancy has been considered by some observers to depend on the chlorosis, so frequently a concomitant or a consequence of the pregnant condition, though it might also be explained by reflex irritation on the middle cerebral arteries originating from the uterus itself. Even in rheumatism, where no cardiac vegetations exist, the choraic symptoms may be set up by the blood being rendered imperfect for nutritive purposes. Dr. Ogle at least considers the disease to be induced in some cases by the anæmia consequent on rheumatism. Still, in this instance we are unable to exclude the possibility of embolism, even where no vegetations have existed in the heart, as Dr. Bastian has found embolisms in chorea composed of agglomerations of white blood corpuscles.

And, once more, the success of certain remedies points to the same thing. We have to encourage collateral circulation in cases of embolism, and better nutrition in those cases in which no definite lesion exists. The mineral tonics seem in many cases to shorten the attack, and sometimes they do so with such rapidity that it is impossible to believe that there has been time for collateral circulation to be set up. The advantage, the necessity, even, of good food in the treatment of such cases, demonstrates the same thing, viz., that we have to do with an instability of a region of the brain due to imperfect nutrition; that this imperfection of nutrition may depend solely on a poor and innutritious blood; but that in many cases we have to do with some coarsish lesion of the corpora striata and the cortical structure of the cerebral convolutions, such as hæmorrhage, softening, tumour, and especially embolism, inducing a species of discharge.

A very uncommon case of congenital chorea has been published by me, and is evidently not an instance of embolism:—

Henry S., aged 13. His mother was frightened at the end of the second month of pregnancy, by squeezing a kitten to death accidentally in moving a barrel, and witnessing its death agony. The child was born about six weeks before the full time. He has had chorea from his birth, and has always been as bad as at present. Lately has learnt to say a few words. Until recently could not speak at all, but could hum tunes in an indistinct way, and make various noises. Cannot

stand or hold anything firmly. His education has been entirely neglected, but he is very sharp and very merry. Can crawl in two ways, and at a great pace, and for this purpose combines his muscular movements pretty well. Is almost always in motion, except when asleep, and then is quite still. Had fits when three years old, and not since. No rheumatism or heart disease; and there is none in his family.

He was put on arsenic. After a few weeks he began to say more words, and was much less restless. Walks rather better, but cannot stand without support. The nurse took great pains with him, and after two months he was able to walk with the help of one hand, and to stand without assistance. He learnt to say a good many words, and proved exceedingly intelligent. Most of his movements showed great deficiency of coördinating power, but he combined movements for the purpose of scuttling along the floor in a wonderful manner, one leg being stretched out in front and the other behind, whilst he paddled along with one hand at great speed. Playing at nine-pins brought him on considerably, and so did the association with and imitation of two non-choreic boys in the ward.

The following quotation from Professor Ferrier's first paper bears upon the causation of chorea:—

‘The movements which are seen in chorea bear an intimate relation to those of epilepsy, and indicate the same centric causation. They are not mere spasms and cramps, but an aimless profusion of movements of considerable complexity, much nearer the purposive movements of health. They are not so much inco-

herences of muscles as incoherences of movements of muscles. There is some method in their madness. Again, they are successions of movements; moreover, they are successions of different movements. I regard the above experiments, in this case also, as an experimental demonstration of the accuracy of the views of Dr. Hughlings Jackson, who places the proximate cause of these movements in an unstable condition of the grey matter of the cerebral convolutions.'

One point remains to be touched upon, viz., the connection of chorea with abnormal mental phenomena. This fact is easily recognised in very acute cases, and, in less degree, can be verified even in ordinary cases of subacute form.

I once admitted into the Bristol Infirmary a girl who had been epileptic for some years. It was about the time of the Ulster revivals, and this girl had been much worked upon by some injudicious ministrations of a young curate. She became rather suddenly choraic in an extreme degree, and at the same time possessed with strange delusions. The devil was always present with her, and she bit me one night severely, under the impression that I was myself his Satanic majesty. The treatment that cured the chorea cured the delusions.

Again, in five cases of chorea recorded by Dr. Handfield Jones, the following points among others were illustrated: (1) the tendency of chorea proper, motor-centre disorder, to be attended with an analogous state of the emotional or of the intellectual centres; and (2) its liability to occur in a modified

form, and to be complicated with quasi-epileptic attacks.

We can easily realise that any lesion that blocks important portions of the middle cerebral artery may gradually induce softening, if collateral circulation is not sufficiently set up, even if no rupture of the artery takes place, and that abnormal mental phenomena may be the result of this lesion. But such a lesion as softening requires some time for its development, and it is not capable of explaining the delusion, the delirium, or the hebetude so often met with in these patients coincidently with the appearance of the jacitation. It is, however, quite easy to believe that the same anæmia, the same want of nutrition, that induces the peculiar motor phenomena of chorea by its action on a special motor centre, may coincidently induce the mental phenomena by extension to the portions of the brain concerned in intellectual function.

And still more easy it is to understand the co-existence of both series of phenomena, now that we know by experiment that irritation of portions of the cortex of the cerebral hemispheres—and these, too, regions hitherto believed to be concerned solely in intellectual work—will produce movements in certain portions of the body. The mental, in fact, own in most cases the same causative lesions as the motor phenomena.

Consult, for Epilepsy—

Russel Reynolds on 'Epilepsy.'

Radcliffe on 'Epilepsy.'

Sieveking on 'Epilepsy.'

- Jaccoud, 'Path. interne.'
 Romberg, 'Dis. of Nervous System.'
 Wilks, 'Path. Anatomy.'
 Trousseau, 'Clin. Lect.'
 Bright's 'Med. Rep.'
 'London Hosp. Rep.,' vol. i. Dr. Hughlings Jackson.
 'West Riding Asylum Rep.,' vol. ii. 304. Dr. Thompson.
 Ibid., vol. iii. 94. Dr. Ferrier.
 Ibid., vol. iii. 326. Dr. Hughlings Jackson.
 'Journal of Mental Science,' April 1873. Dr. Crichton Browne.
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Consult, for Chorea—

- Dr. West on 'Diseases of Infancy and Childhood.'
 Todd, 'Clinical Lectures.'
 Reynolds, 'Syst. of Med.' Dr. Radcliffe.
 Jaccoud, 'Path. interne.'
 Romberg, 'Dis. of Nervous System.'
 Hammond, 'Dis. of Nervous System.'
 Wilks, 'Path. Anatomy.'
 Bright's 'Med. Rep.'
 'Med.-Chir. Review,' lxxxii. Dr. Ogle.
 'London Hosp. Rep.,' vol. i. Dr. Hughlings Jackson.
 'Hemichorea.' Pamphlet. Dr. Hughlings Jackson.
 'Med. Record,' i. 263. Dr. Foot.
 'New Syd. Soc. Yearbook,' 1867. Roger.
 Ibid., 1871. Murchison and Handfield Jones.
 'West Riding Asylum Rep.,' vol. iii. Dr. Ferrier.

LECTURE XII.

TETANUS.

A RECENT writer has said, with reference to the pathological anatomy of tetanus, that the lesions have nothing that marks them as exclusively peculiar to the disease ; whilst the instantaneous development of the tetanic phenomena and the possibility of the recovery scarcely allow us to assign a primary character to these lesions. There are, it is true, some secondary alterations, as Rokitansky and Demne have proved ; one of the facts recorded by this latter observer proves this view very satisfactorily, for the lesion was followed up from the centripetal nerves to the posterior columns ; it consisted in proliferation of the neuroglia amounting to sclerosis, and was distributed sometimes uniformly over a certain extent, sometimes in spots irregularly disseminated. To these alterations, which have been thoroughly recognised both in themselves and in their relation with tetanus ever since the works of Rokitansky, Demne, and Wagner, it may be well to add the granular degeneration of the cells of the cord, discovered more recently by Lockhart Clarke. According to him this latter lesion is constant, although this is a point that further observation ought to clear up ;

but the alterations of the neuroglia may be wholly absent, as Leyden's facts prove. The older authors have indicated a great number of other disorders of the nervous centres, but these are accidental and accessory lesions, without any certain relations with tetanus. A single exception may be made for an alteration which is not more constant than those above-mentioned, but we can have no doubt it bears some relation to the disease, viz., inflammation of the neurilemma mentioned by Lepelletier and Froriep, and traced by them from the nerves near the wound to the cord.

In a majority of cases in which the spinal cord has been examined, both the cord itself and its membranes have been found healthy; in others there has been found a collection of fat between the membranes and the cord from the lower part of the cervical to the middle of the dorsal region; in one there was a small softened patch in the cord just above the cauda equina; in another there was an effusion of blood outside the dura mater about the 7th or 8th dorsal vertebra, and a softening of cord in the dorsal region; in this case there had been a blow on the back. In the case of Cook, poisoned by Palmer with strychnia, the prisoner in great measure grounded his defence on the presence of a few granules on the cord, the result, probably, of slight antecedent meningitis. Effusions of serum into the spinal membranes and even hæmorrhages are not very uncommon in tetanus, and are results and not causes of the phenomena. I shall refer directly to lesions found by Lockhart Clarke, Dickenson, Allbutt, and others, in speaking of the microscopical appear-

ances of my own cases. The nerve in the wound in traumatic tetanus has been found diseased. Mr. Erichsen says that the twig in the wound is generally diseased. In thirteen cases recorded by Mr. Poland in Guy's Hospital reports the nerve twig was found inflamed in five and bulbous in one. Trousseau also gives some instances. In those cases, moreover, where the disease has been arrested by releasing a nerve-fibre which had been included in a ligature, or by excising the injured part, and thus isolating the termination of the nerve, we must receive some morbid condition of the nerve itself in the wound as an explanation of the exciting cause of the disease. Against the universality of this view, however, the nerve is often found inflamed and otherwise diseased without the occurrence of tetanus; and again, in a large majority of cases of traumatic tetanus we can recognise no morbid condition of the nerve.

If, therefore, we are compelled to exclude the condition of the nerve as causing the tonic spasm, we must look to portions of the cerebro-spinal system or to the blood. The vaso-motor system is necessarily excluded, because the lesions found in the ganglia are met with only exceptionally, although, probably, the sensitiveness of these nerves to reflex impressions is one of the primary phenomena in the attack. What, then, causes this kind of spasm?

Dr. Roemer, referring to certain experiments of Dr. Weir Mitchell on the cerebro-spinal fluid, and its agency in producing convulsions under pressure, says: 'On injecting half an ounce of water (66° Fahr.) into the spinal canal of a rabbit, convulsions ensued, and shortly afterwards death. By this injection the blood

was displaced in the spinal vessels, and caused bleeding from the exposed veins of the cord and head. A larger amount of water under 100° Fahr. was borne in the second experiment, and spasms followed instantly upon the introduction of water at 32° Fahr. Every variety of convulsive action was thus effected. Subsequent experiments proved that the local and external application of extreme cold upon the spine brought about similar results. The agent usually employed was rhigolene. It results from these experiments that at or about the 14th vertebra from above downwards we cease to notice backward spasm and stupor, and see only signs of weakness or of tetanic rigidity in the legs. A jet of rhigolene thrown upon the spine of a frog occasions spasmodic movements of the legs, and at intervals violent tetanic contractions. The pigeon, under the application of cold to the side, may be handled or laid on its back and side, while at any moment a loud sound or a sudden motion will break the spell, and it will abruptly run backwards several feet. The following symptoms of partial tetanus (*emprothotonos* and *opisthotonos*) should be marked. In the spasm from chilled spine or cerebellum the head is carried at ease during the interval between the fits, but at the moment of attack the bill strikes the floor quickly, first on one side and then on the other, the head being drawn violently forward. Even in the most terrible of the somersaults caused by cold, the head was drawn forward, and the backward turn was produced by the action of the muscles of the legs and wings, rather than by those of the back, neck, and spine. Very little of the convulsive acts is

due to the direct effect of cold, much more to the intense and overpowering congestion, which in time wears off. The added proof lies in the fact that local irritants, which congest more slowly, occasion in the spine the same phenomena after the lapse of a longer interval. Intense cold to freezing and the injection of water produced consequently similar effects, that is, any displacing agency upon the spinal fluid gives tonic spasm. Direct injury to the cerebellum and spinal cord conditions similar results. Fodère, Majendie, Flourens, Purkinje, and Krauss have observed tonic spasm and opisthotonic movements in mammals and birds after the loss of the cerebellum. Mitchell also has verified these experiments.

Besides this, Professor Ferrier has shown that powerful irritation of one corpus striatum causes rigid pleurosthotonos, the flexors predominating over the extensors; whilst the optic lobes, or corpora quadrigemina, besides being concerned with vision and the movements of the iris, are centres for the extensor muscles of the head, trunk, and leg, and irritation of these centres causes rigid opisthotonos and trismus. Again, it seems proved that sudden arterial contraction in the spinal cord, the medulla oblongata, and probably in the meso-cephale, induces tonic spasm, of which tetanic spasm is so far a mere variety that in it one set of muscles overcomes the resistance of their opponents; the flexors, for instance, being more strongly affected than the extensors.

As in epilepsy, this impressibility of the vaso-motor nerves that rule the calibre of the arteries must be produced in some way. Is it due to a lesion of a seg-

ment of the cord with which the vaso-motor nerves are associated, or to some alteration in the blood itself? The condition of the nerve in the wound, if it were true that the nerve was often found diseased, would be necessarily unequal to the production of the tetanic state, unless the spinal cord or its extensions in the brain, the vaso-motor nerves themselves or their ganglia, were already abnormally impressible. The nerve in the wound may be the channel by which the excitation is carried to the cord, the state of the nerve may even be the exciting cause of the spasm, but there is something over and above this without which the exciting cause would fail to produce the tetanic phenomena. Besides, tetanus is not only traumatic, but idiopathic, set up by a variety of causes, cold, damp, bad ventilation (especially in the case of infants), sudden changes of temperature, &c. Here the state of the eisdic nerves shows no abnormality.

We begin, then, with the lesions of the cord itself. Now that minute investigation is more frequently made at autopsies on patients who have died of this disease, it is more common to find some lesion or other of the cord and its membranes.

We have to consider : (1) What these lesions of the cord are. (2) Are they the cause of the phenomena, or their effect? (3) Are they met with in the cord under circumstances wholly unconnected with tetanus?

Romberg tells us that we may find congestion, inflammation, exudation, softening, induration, but that the appearances are very inconsistent ; and Dr. Wilks, that the affection of the nervous system is able to kill

without leaving any traces behind, and thus, in general terms, the body is healthy. In a case of idiopathic tetanus, however, Romberg found that the density of the medulla oblongata presented a marked contrast to the great softening of the cervical portion of the cord, which exuded from its membranes on slight pressure. The dorsal portion was firm ; the lumbar portion, again, almost liquid.

Sandras and Bourguignon say that one meets with some lesions of the spinal membranes, or a little more liquid than usual in the meninges, or injection and even softening of the anterior columns of the cord. But the former lesions, such as cartilaginous plates, thickening, &c., belong to organic affections generally much older than the commencement of the tetanus. On the one hand, the increase of the fluid is not always there, and, on the other hand, it is met with in a number of diseases which have nothing in common with tetanus ; and, lastly, it may reasonably be asked if this lesion is not the effect of the malady rather than the cause.

May we not say the same of hæmorrhagic injection of the grey and white substance of the cord, and of softening of the anterior columns ? Doubtless, when in the bodies of persons who have died of tetanus we meet with a decided hæmorrhagic injection of the white or grey substance of the brain or cord, we are justified in saying that we have got one notion more on the anatomy of the disease. ‘*Mais cette notion est-elle applicable à la cause ou à l’effet ? Que deviendraient les cas dans lesquels on ne trouve rien de semblable ?*’

For it is certain that these lesions, sometimes remarked, are not invariably found; many authors do not speak of them as being constant. And these observers go on to say that in death from strychnia the amount of the cerebro-spinal fluid and the redness of the grey substance are not constant appearances, and when they are met with they seem to be in direct ratio with the *gradual* approach of the fatal event. 'It is clear,' says Dr. Handfield Jones, 'that in all cases a certain predisposition of the cord must exist before the spasmodic symptoms declare themselves.' As Dr. Watson says, the real mystery lies in this predisposition.

The first and most important observations made in England, microscopically, on the spinal cord in tetanus were by Lockhart Clarke. In 1865 he contributed six cases to the Medical Chirurgical Society, in all which areas of disintegration were found in the spinal cord. The first case was reported at some length, and the lesion was found more or less from the origin of the second cervical nerves to the lumbar enlargement. At the second cervical nerves, streaks and irregular areas of disintegration were observed in different parts of the grey substance, and particularly around the central canal, on the right side of which was a space of considerable size, containing a finely granular fluid, with the débris of blood-vessels and nerves. The posterior and lateral white columns, especially along the edges of the various fissures which transmit blood-vessels, were damaged in a similar way; and in some sections the deeper portions of the posterior columns which rest on the transverse commissure were softened

to a considerable degree. This disintegration was still more marked in the cervical enlargement, chiefly behind and at the sides of the canal. The posterior commissure was wholly and the anterior partially destroyed by a fluid, transparent, and granular area. Throughout the cervical enlargement similar lesions were discovered, varying from a state of softening to a state of complete solution, and diminishing at intervals or almost disappearing, to return shortly in the same form. At the upper part of the dorsal region the shape of the cord was much altered, and extensive lesions of the same kind were everywhere seen. In both lateral halves of the grey substance, the left lateral column, the right antero-lateral column, the superficial portion of the anterior columns, and in the posterior columns similar appearances were found. Below this point there was less disease as far as the fourth dorsal vertebra. Here, in addition to the areas of disintegration, large extravasations of blood were found along the whole lateral part of the grey substance, on both sides in some sections, on one side only in others; whilst the lumbar regions manifested the same lesions as the cervical. In the second case, which was one of idiopathic tetanus, areas of disintegration and exudation were discovered in different parts of the cord, but chiefly in the central grey substance. In the other cases similar conditions were found, but not always over the same extent of surface.

Dickenson describes somewhat similar lesions in a case of traumatic tetanus, attacking the central grey matter specially. The cord seemed to the naked eye

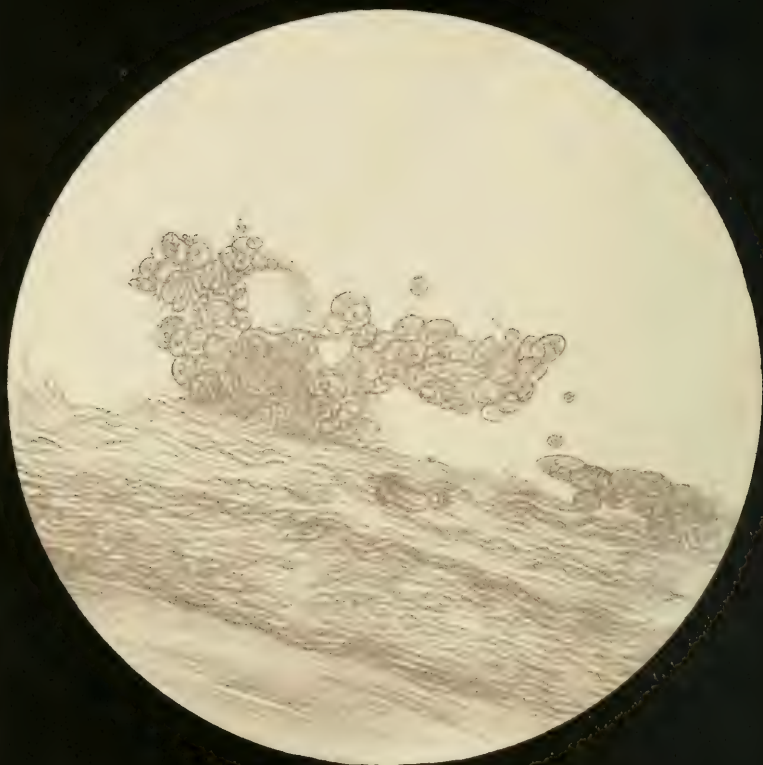
the seat of three swellings. On the right side of the cervical enlargement was an oval swelling about the size and shape of a split bean, which lay between the roots of the nerves. A more extensive change of the same kind was found opposite the first lumbar vertebra; and about half-way between this tumefaction and the lower end of the cord was a smaller circumscribed prominence of the same nature, which lay likewise on the posterior aspect of the cord. There was great injection of the spinal dura mater and pia mater. In this case the blood-vessels appeared to be, if not the first, at least an early seat of change. Distended with blood, not only to the uttermost of their natural capacity, but dilated to many times their proper width, and crammed with blood corpuscles so as to look like solid cylinders, their condition gave evidence of an altogether abnormal relation between the pressure of the blood and the resistance of the walls. Either blood had been propelled into them with supernatural force, or, what is more likely, the tension of their coats had been lessened by a change in their innervation.

The overcharge of the vessels led to the escape of their contents. In some places blood corpuscles were extended. More often only the fluid portions of the blood traversed the walls, to appear as the translucent structureless material which played so prominent a part in the destruction of the cord. That this translucent material was an exudation from the vessels, and not a product of disintegration of tissue, is evident from the following facts. It constantly lay in contact with the vessels and often surrounded them. It held

the most changeable relations to the nervous matter, lying in the grey matter, in the white, abundantly in the fissures, and occasionally outside the cord altogether. It was the source of increase of bulk, of laceration, and of displacement, such as to suggest that it was an addition to the structure, and had been forcibly driven into it. At the same time a certain amount of disintegration of the nervous elements had taken place where the exudation came in contact with them; and this tendency to disintegration in the nervous matter may have been enhanced by the unnatural state of the blood-vessels, and the consequent imperfect nutrition of the cord. The distension and repletion of the blood-vessels throughout the cord involved both arteries and veins, and, in a less degree, the capillaries.

Dr. Clifford Allbutt has published notes of four cases of tetanus, with an examination of the spinal cord in each. The lesions were diminution of consistence of various degrees and situations in the cord, hæmorrhage in two of them, visible to the naked eye. On microscopical examination, there was great distension of the blood-vessels in both white and grey matter, with occasional exudation and disintegration of tissue around them; isolated patches of disintegration of various shapes and sizes in both grey and white matter; and in the grey matter numerous vacuities, having on transverse section circular or oval outlines, and resulting from disintegration of the nerve-fibres.

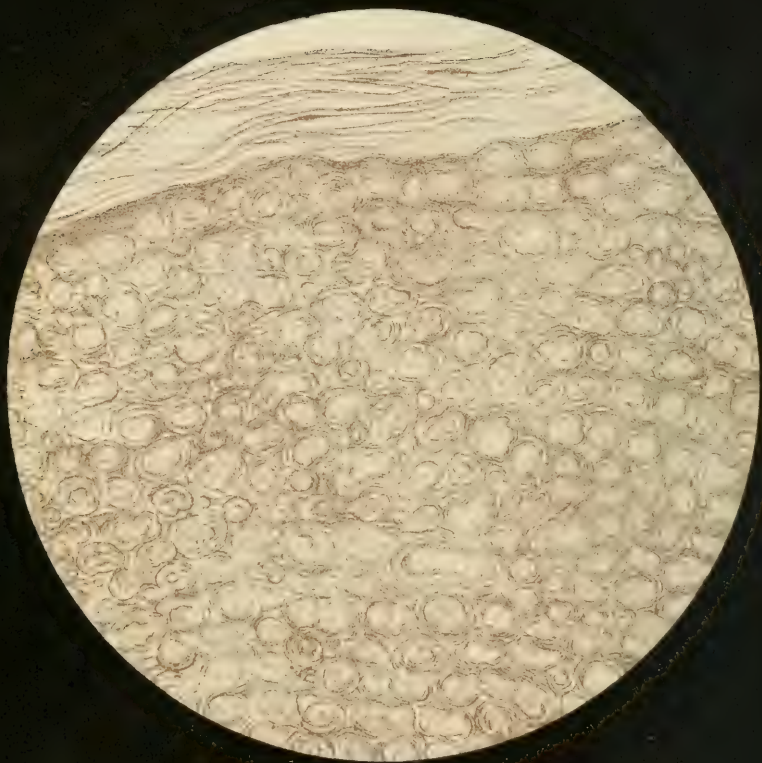
In my own cases, in which minute examination was made, the appearances varied very much.



C. Berjeau, Lith

Banks & Co., Print

TETANUS
(NEW MATTER ON DURA MATER)



TETANUS
(COLLOID DEGENERATION)

In case 1, the only abnormality to be remarked was dilatation and distension of the vessels of the spinal pia mater. The cord itself was healthy.

Case 2, Plate 17. Man, aged 36. Brain quite healthy, except that the central white matter round the ventricles and the crura cerebri were rather soft. Spinal cord universally soft in its whole extent and thickness. About the 6th or 7th cervical vertebra this softening of the whole thickness of the cord amounted to a diffuent condition; and the cord here under the microscope seemed composed of broken-down nerve-fibres; nerve-ganglia, and oil. At this point also the dura mater was rather adherent to the bodies of the vertebræ, and beneath it was poured out some new material.

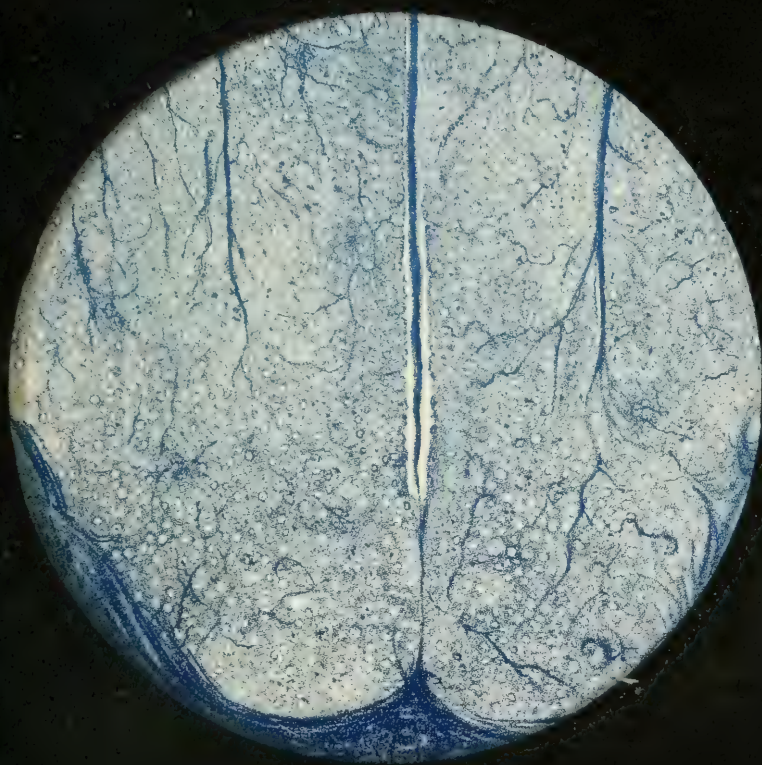
Case 3. Boy, aged 15. The spinal cord and membranes were healthy down to a spot corresponding to the 10th or 11th dorsal vertebra. Here there was a small quantity of blood effused outside the spinal dura mater; a little gummy-looking fluid existed beneath the arachnoid. The spinal cord at one spot here was much softened, almost diffuent, and was ruptured on the very gentlest traction. This condition existed through its whole thickness for about half an inch at this spot. There were many amyloid bodies in the grey substance and some thickening of the vessels.

Case 4, Plate 18. Boy, aged 9. A good deal of blood existed in the external veins of the cerebral hemispheres. Brain healthy, cerebellum a little soft, as was also the posterior portion of the medulla oblongata. The posterior columns of the spinal cord throughout their whole length were extremely softened,

almost resembling cream. This was especially apparent immediately below the medulla oblongata, and about the middle of the dorsal region of the cord. This softening was entirely limited to the posterior columns, except at the very top of the cord, where for about an inch the whole cord seemed almost equally soft. In all other parts the rest of the thickness of the cord was firm and healthy. The softened portion was very white, not reddened at any spot; and was composed of an immense number of globular bodies, somewhat irregular in size and shape, 'colloid degeneration of the neuroglia nuclei,' and no trace of the structure of the cord remained.

Case 5, Plate 19. This plate was from a case of tetanus of which I have not the history. It is very little magnified, but shows very well the white spots of colloid degeneration, the dark dots of amyloid bodies, and thickening of vessels. The portion of cord represented is that between the posterior horns of the grey matter.

Here then are a considerable number of instances in which the spinal cord has been examined microscopically and lesions found; lesions, it is true, varying in nature, but having pretty much the same significance. It is tolerably certain that these lesions do not exist in cases of recovery; or, if they do, they must be of extremely small extent. We may be sure that hæmorrhage has not taken place if a tetanic patient recovers without any trace of paralysis being left. Grave degenerations of the cells of the neuroglia, amyloid, or colloid, would be conditions wholly irrecoverable, as



x 20

C. Dreyer, lith.

Barber & Co., Edinb.

TETANUS

(CEREBELLUM AND AMYLOID DEGENERATION, THICKENED VESSELS)

far as we know. Again, when the exudation, either by pressure or by some solvent action, has caused grave and extensive disintegration of portions of the spinal cord, it is highly improbable that recovery can take place. As, however, a certain number do get well, especially of idiopathic tetanus, we may be sure that these severe lesions do not obtain in all cases. The distension of the vessels and a certain amount of exudation can be got rid of, and the cord become almost, if not completely, the same as before.

Lockhart Clarke asks ‘Are the structural lesions or the disintegrations of tissue the effects of the functional excitement of the cord manifested in the tetanic spasms?’ That they are not the effects of this excitement would appear, he thinks, from the following facts: ‘That they more frequently occur in the central parts of the grey substance around the canal, where the nerve-cells are scanty; that the nerve-cells of the anterior grey substance, which give origin to the motor nerve-roots, remain apparently unimpaired; that the structural changes commence frequently in company with exudations around or in the vicinity of blood-vessels which are themselves commonly found dilated and frequently in a state of disintegration; and lastly, that they are exactly similar in kind to the lesions and disintegrations which I find in various cases of ordinary paralysis, in which there is little or no spasmodic movement. It appears, therefore, that they result from a morbid state of the blood-vessels, and not from excessive functional activity of the cord.’

Still, though the whole of the lesions may not be

the effects of the spasms, some may be. The distension of the vessels is not unlike what is often seen in the medulla oblongata in epilepsy, the dilatation having occurred in consequence of the primary arterial contraction. It is not impossible that the exudation may have been the consequence of this same over-distension, the congested vessels relieving themselves under considerable pressure. Much of the disintegration may be due to the mechanical pressure of this very exudation, whilst some of it may own as its cause the impaired nutrition consequent on the frequently repeated contraction of the arteries. The amyloid and colloid degeneration of the neuroglia cells are met with under circumstances both of impaired nutrition and of slow inflammatory action.

The third question, as to whether the same lesion may be found under circumstances wholly unconnected with tetanus, has been practically answered already. There is no one lesion of those above-mentioned that may not be found in cases of ordinary paralysis, in progressive muscular atrophy, in locomotor ataxy, &c; only in these latter diseases the lesion is more or less strictly confined to a particular district of the spinal cord. In such diseases there is nothing analogous to tetanic spasm.

Thus far we find the pathological anatomy of tetanus in this position : 1. Lesions of the peripheral end of the nerve in a certain proportion of cases of traumatic tetanus; such lesion being absent in many cases, and of course not present in instances of idiopathic tetanus. 2. There are many and various lesions of the spinal

cord frequently met with, involving both grey and white matter, but especially the commissures of the cord and the immediate neighbourhood of the blood-vessels; not only, however, may tetanus exist without any of these spinal cord lesions, but the abnormalities of the cord may be present without tetanus. These lesions may, in fact, be effects of the tetanic spasm, or of the arterial contraction which is one of the first phenomena of the attack; but they do not enter into the causation of the tetanus further than in this limited way, that any malnutrition of the cord may render it abnormally impressible.

What then is the cause of the tetanic phenomena?

No doubt sudden variation in temperature, bad ventilation, cold and damp, may all be predisposing causes. But how? Is there one cause for traumatic and another for idiopathic tetanus; or is the chief difference this, that the primary lesion is external in the one case and internal in the other?

Lockhart Clarke attempts to answer the question thus: 'Since lesions of the cord in cases of paralysis, in which there is commonly no spasm, are similar to those of tetanus, it follows that the latter disease, in regard to its morbid anatomy, differs from the former only in being associated with a morbid condition or injury of some of the peripheral nerves. It would therefore appear that this condition or injury of the peripheral nerves is the determining cause of the phenomena, and that the spasms of tetanus depend on the conjoint operation of two separate causes. First, that they depend on an abnormally excitable state of the grey

nerve-tissue of the cord, induced by the hyperæmia and morbid state of its blood-vessels, with the exudations and disintegrations resulting therefrom. This state of the cord may be either an extension of a similar state along the injured nerves from the periphery, or may result from reflex action on its blood-vessels excited by those nerves. Secondly, that the spasms depend upon the persistent irritation of the peripheral nerves, by which the exalted excitability of the cord is aroused; and thus the cause, which at first induced in the cord its morbid susceptibility to reflex action, is the same which is subsequently the source of that irritation by which the reflex action is excited; whilst in idiopathic tetanus arising from exposure to cold and damp it is probable that the morbid condition of the blood-vessels of the cord results from change in the state of the peripheral nerves, which may act through reflex actions or otherwise.'

The objections to this view are: 1. That there is often no morbid condition of the nerve at all, or at least no other abnormality than is constantly seen after operation without tetanus; 2. That it is improbable that hyperæmia of the cord can be induced by an extension of a similar state along the injured nerve from the periphery, whilst reflex action from the injured nerve would induce contraction of arteries rather than hyperæmia; 3. It affords no explanation for those cases in which no spinal cord lesion exists.

I believe that in tetanus we must look for the connective lesion in the chemistry of the blood. In strychnia-poisoning, a disease so closely analogous to

tetanus, the presence of strychnia in the blood, or rather the effect that strychnia has on the physiological conditions of the blood, has been demonstrated as the cause of the spasmodic phenomena. Dr. Harley has shown that blood mingled with a solution of strychnia is incapable of absorbing oxygen in the usual proportion.

With symptoms almost precisely similar, it is difficult to believe that tetanus is a functional disease, acknowledging as its cause no necessary organic change and no abnormality in the blood. And as death may occur from strychnia, independently of asphyxia, or inanition, or exhaustion, by destroying the chemical power of the blood for assimilating oxygen, and thus rendering muscular respiration impossible, so cases do occur in traumatic tetanus where death ensues while the patient is taking plenty of nourishment and stimulants, where there is no bar to the pulmonary circulation, and with too little spasm to cause exhaustion, and the patient sinks as surely from a poisoned condition of blood, a blood unfit for the purposes of life, as in those cases of cholera well-nigh instantaneously fatal which occur occasionally in the course of an epidemic. The abnormal blood may or may not lead to the various lesions of the cord which have been already mentioned. It must, however, from the first, modify decidedly the nutrition of the cord. If this nutrition is not modified to too great an extent, and especially if the organic lesions dependent on such modifications of nutrition are not developed, the patient recovers, if the blood is gradually restored to its power of taking up oxygen.

It is very suggestive, especially when we consider the distended condition of the vessels in fatal cases, that the remedies most useful in tetanus are those which partially paralyse the vaso-motor nerves and thereby distend the vessels. I feel sure that prolonged contraction of the arteries is the phenomenon to be most dreaded, and that the hyperæmia found after death is really the reaction from this previous state of contraction.

The abnormal blood imperfectly nourishes the cord; an imperfectly nourished cord is *ipso facto* an excitable, an impressible cord; this impressibility renders arterial spasm abnormally facile, whether the exciting cause is the circulation in the cord of more of the morbid blood, or reflected irritation from a diseased nerve at the periphery, or reflex irritation from any other cause and from any other point in the body; and if this arterial contraction goes on for any protracted period, or is frequently repeated, we may find various lesions due to imperfect blood-supply in addition to those due to diminished nutrition from the original nature of the blood, whilst as a sequence of the spasmodic arterial contractions we get hyperæmia and perhaps exudation; and lastly, the pressure of the exudation, or some peculiarity in its nature, may lead to further disintegration of the nervous centre.

It is right to say a few words on tetany or tetanilla, as both in its symptoms and its pathology it seems somewhat connected with tetanus. It is characterised by intermittent paroxysms of tonic spasms affecting the upper and lower limbs, and in

severe cases nearly all the muscles of the body. The spasm is ushered in by a sensation of tingling, followed by a feeling of hesitation in movement, and is attended often by considerable pain, and by no loss of consciousness.

Trousseau thinks it can be diagnosed by the appearance of the hand during a paroxysm; the thumb is forcibly and violently adducted, the fingers are pressed together closely, and semiflexed over the thumb in consequence of the flexion of the metacarpophalangeal articulation, and the palm of the hand being made hollow by the approximation of its outer and inner margins, the hand assumes a conical shape, or rather the shape which the accoucheur gives to it when introducing it into the vagina.

It is diagnosed from tetanus, in that in tetanus the muscles of the jaws are first affected, then of the face, and then the trunk, whilst the spasm only by degrees extends to the extremities; but in tetany the contractions run an opposite course, and it rarely happens that the muscles of the extremities and those of the rest of the body are affected at the same time.

The disease is seldom fatal, and pathological anatomy has done very little in clearing up the nature of the complaint; but, as Trousseau says, 'from a mere review of the symptoms it is impossible to admit that such mobile and transitory phenomena can be due to the existence of serious organic lesions.' He considers it to be due to rheumatism.

Hydrophobia.—So little has been known hitherto of the pathological anatomy of this disease, that many

writers avoid all mention of it in their works, and others are content to dismiss it in a few words.

Morgagni, Mead, and Van Swieten affirm that there is nothing abnormal in patients who have died of this malady. Others only make the remark that there are some traces of asphyxia, some engorgement of the lungs, a venosity of the blood, and sometimes an effusion of serum in the ventricles and in the meninges of the brain and cord, a condition that might well be due to the asphyxia. Trousseau says that the pathological changes found after death are only those dependent on the asphyxia which occurs in the last stage.

Hyperæmia of all the parenchymatous organs is met with as a consequence of the final convulsion.

The spleen is sometimes found abnormally enlarged.

Rudnew holds that hydrophobia, like almost all other infectious diseases, is connected with a profound disturbance of all the important organs. In all his cases the kidneys manifested a highly developed parenchymatous inflammation, the peculiarity of which was that both cortical and pyramidal portions were alike affected, presenting all the conditions necessary for the production of uræmia. He thinks it extremely probable that the latter may be the cause of many of the symptoms. A second peculiarity was the degenerative character of the nephritis; in the most advanced and fatal stage of the disease the urinary tubules were completely bare of epithelium, and filled with a granular fatty degenerated material.

In Dr. Wilks' fatal cases the body was livid, particularly the face, which looked like strangulation. The blood was of a dark colour and quite fluid, both in the vessels and in the heart; the lungs were much congested, and the posterior parts of a very dark colour and soft; the bronchial tubes also congested and of a dark colour. The kidneys, liver, &c., all congested; and the brain and spinal cord showed no other appearance than congestion, due to the mode of death. The larynx was healthy, but the pharynx presented a very unusual condition, being dilated, as if violent muscular contraction had occurred in it; the mucous membrane was swollen; the glands were enlarged and covered with thick secretion, as were all the glands at the back of the tongue.

In the beginning of this century a Russian physician, Dr. Marochetti, in a memoir on hydrophobia, and Dr. Xanthos of Siphnos, in a letter to Hufeland, called attention to the presence, on the under surface of the tongue, near the frænum, of pustules or vesicles of a special character during the stage of the incubation of rabies. These had long been known in Greece under the name of 'lyssi.' The presence of these pustules had long been known also traditionally in Russia and in Greece. These physicians believed that, if these vesicles or pustules were laid open in time and cauterised, all manifestations of rabies were prevented. These lyssi, however, have not been found in all cases.

Cases of hydrophobia are happily not very common, and in many instances observers have been content with recording naked-eye appearances. To Dr. Clifford

Allbutt, however, the profession are indebted for careful observation of two cases. In one only the medulla oblongata, the pons, and the spinal cord were examined ; in the other the whole of the cerebro-spinal centres. Throughout all these centres the same morbid conditions were observed. There was visible great vascular congestion, with transudation into the surrounding tissues. In the cerebral convolutions, the meso-cephale, the pons, medulla, and spinal cord, the vessels were seen in various degrees of distension ; in many places their walls were obviously thickened, and here and there in them were patches of incipient nuclear proliferation. On removing the parts there was found in many places a diminished consistence, and this was well marked in the medulla.

This seemed to be due to serous infiltration with soddening, and was seen in the convolutions, in a few parts of the central ganglia, in the medulla in both cases, and here and there in the cord. In both cases, too, there seemed to have been a transudation of something more than serum. In some sections large hæmorrhages were visible ; in some few other places minute hæmorrhage had occurred, and in many spots there was a refracting material outside the vessels, which probably was of the nature of a coagulated fibrinous exudation. Finally, there were found in the encephalon, occasionally, in both spinal cords more frequently, and in both medullas abundantly, little gaps caused by the disappearance of nerve-strands, which had passed through the granular disintegration of Clarke. These phenomena, together with the enlarged spleen found in both cases,

point to the action of an animal poison acting primarily on the cerebro-spinal nervous system. The order of the severity in the various parts seemed to be—first and worst, the medulla; secondly, the cord; thirdly, the cerebral convolutions; and fourthly, the central ganglia of the encephalon.

These lesions would be of still higher importance if they occurred in all cases; but Dr. Elder has recorded three cases of hydrophobia in which no lesion was found in the cerebro-spinal nervous system, and in one of these the cerebellum, the medulla oblongata, and part of the cerebrum had been microscopically examined by Lockhart Clarke himself, who reported that they were free from all lesion, except congestion.

Abercromby mentions that in hydrophobia inflammation of the cord had once been remarked.

Besides, then, the morbid phenomena due to the mode of death, such as engorgement of the lungs and other viscera, congestion of brain and spinal cord, &c., there have been at present recognised a peculiar development of the glands of the fauces, vesicles, or pustules under the tongue, and these especially during the period of incubation of the disease, a special degenerative nephritis, an enlargement of the spleen, and granular disintegration of the convolutions, the medulla oblongata, and the spinal cord; but at present we cannot say that there is any definite lesion peculiar to the disease, and certain to be found post mortem.

Consult, for Tetanus—

Jaccoud, 'Path. Interne.'

Reynolds, 'System of Medicine.'

- Wilks, 'Path. Anatomy.'
Rokitansky, 'Path. Anatomy.'
Romberg, 'Dis. of Nervous System.'
Trousseau, 'Clinical Lectures.'
Sandras & Bourguignon, 'Maladies Nerveuses.'
Handfield Jones, 'Functional Dis. of Nervous System.'
Bright's 'Med. Reports.'
'New Syd. Soc. Yearbook,' 1871-72. Dr. Allbutt.
'Med.-Chir. Review,' 84, 86. Dr. Ogle.
'Guy's Hosp. Rep.,' xv. Mr. Poland.
'Med.-Chir. Trans.,' 51. Dr. Dickenson.
'Med.-Chir. Trans.,' 48. Lockhart Clarke.
'Med. Record,' 1, 533. Roemer.

Consult, for Hydrophobia—

- Wilks, 'Path. Anatomy.'
Trousseau, 'Clin. Lectures.'
'New Syd. Soc. Yearbook,' 1871-72. Rudnew.
'Brit. Med. Jour.,' vol. ii. 1871. Dr. Elder.
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'Med.-Chir. Review,' July, 1874.

LECTURE XIII.

LESIONS OF THE SPINAL CORD IN SMALL-POX.

SOME few cases have been observed and the cords examined by Westphal, Lockhart Clarke, and others, where, in the course of an attack of variola, certain paralytic phenomena had manifested themselves. In Westphal's first case the patient felt in the third day of discrete small-pox some weakness of the legs. Complete paralysis supervened, so that he only retained some slight power of moving his great toes ; reflex movements were abolished ; sensibility unimpaired ; there was paralysis of bladder and a slough over the sacrum ; faradic irritability of the muscles was retained. In the second case motor paralysis of the left leg, with a sensation of numbness in it, was experienced on the eleventh day of the disease. The day afterwards the right leg became paralysed, with incontinence of fæces and a peculiar sensation as if the abdomen were dead. The patient died of cystitis and slough over the sacrum. At the necropsy the grey substance of the spinal cord was found congested, but there was no alteration in either the white columns or in the nerve-roots. In one of the sciatic nerves a slight infiltration of blood was noticed between

its bundles. On making thin sections of the cord, after it had been treated with bichromate of potash, patches variously altered in colour were found to be irregularly scattered through the grey and white substance. Areas of softening, about the size of a pin's head, were seen in the grey substance at the superior thoracic region. In all those places where the colour of the tissue was modified there was an abundance of fatty granulations. Westphal proposes to name this condition 'Disseminated Myelitis.' Vulpian gives a very interesting case in which life was preserved and partial recovery had taken place from a state of partial paralysis of the upper extremities supervening on small-pox. Vulpian thinks that in each case during the development of small-pox there was an inflammatory irritation of the spinal cord in the region corresponding to the roots of the nerves which supplied the muscles and skin of the shoulders. The inflammation probably attacked different elements of the grey substance, and amongst others the nerve-cells of the anterior horns. Then the nerve-fibres of the anterior roots, at least those in connection with the altered cells, underwent granular degeneration, which takes place when the normal influence which these cells have on the nerve-fibres ceases. The muscles supplied by these nerve-fibres became atrophied, and their contractibility was so weakened that it became no longer excitable through the skin by the induced current. This is the necessary result of the lesion which is supposed to have existed here. There was therefore, probably, in this case a group of lesions connected with each other, which

remind us of the group that exists in the atrophic paralysis of infancy. M. Vulpian says that in several cases of small-pox in which there was severe rachialgia he has not found after death any change whatever in the grey or the white substance of the cord.

Lockhart Clarke, who reports these observations of Westphal and Vulpian, published a case of extreme muscular atrophy of the arms and shoulders consequent on inoculation with the virus of small-pox, with extensive disease of the cervical portion of the spinal cord.

We give this case in Lockhart Clarke's own words. The man, aged 32, had been inoculated when about a year old. Before this operation he was a fine healthy child. He suffered much after the inoculation, and gave evidence of severe indisposition; and when the eruption was fully developed, the left arm was observed to droop and become powerless. Soon afterwards the right arm followed the same course. From this period the muscles of the arms and shoulders ceased to be developed, and at the same time the heart's action became irregular and disturbed, and continued so ever after. When he was examined, a few months before his death, the muscles of the shoulders and arms had almost entirely disappeared. The muscles of the forearm were apparently unaffected. The muscles of the ball of each thumb were almost altogether gone, while those of the fingers were well developed. The muscles of the dorsum of each scapula were nearly gone; all those of the lower extremities were well developed. On examination of the cervical enlargement

of the spinal cord after death, I found extensive lesions and displacements of the grey substance. On the right side the posterior horn was drawn backward with a long streak towards the softened surface of the corresponding white column. The cervix cornu on each side was more or less damaged in different sections by a streak of disintegration, and in the anterior grey substance there was an extensive area of softening and disintegration. On the right side the lesions in the central part of the anterior grey substance were less conspicuous, but there was a dilated blood-vessel with its branches bordered by a thin layer of exudation, and by the surrounding tissues in a state of disintegration. Besides other changes in different parts of the cervical region, a great number of the nerve-cells were found to be in different states of atrophy and disintegration. In many instances, the large cells of the anterior cornu were reduced to at least one-eighth or one-tenth of their ordinary size, and some even to mere granular points; while in other instances, although not much reduced in size, they had lost their sharpness of outline, and consisted of an uneven aggregation of granules.

LESIONS OF THE NERVOUS SYSTEM IN DIABETES.

It is probable that the lesions of the nervous system in diabetes are of a twofold nature—the one primary and causative, the other secondary and dependent on the altered nutrition induced either by the presence of an abnormal amount of sugar in the blood, or of the negative loss to the blood in this disease.

The first series of lesions can be produced artificially. If the floor of the fourth ventricle is punctured in a very limited space, the production of sugar is increased, and the animal becomes diabetic. This spot is comprised between the origin of the vagi and the auditory nerves. Bernard found that the channel of communication between this portion of the nervous centre and the liver was not by the pneumogastric nerves. It was proved by his experiments that the superior parts of the spinal cord could be alone concerned; for on going beyond the first dorsal vertebra he failed to produce the phenomenon. The excitement is therefore transmitted by the spinal cord as far as the top of the first pair of spinal nerves, and from this point it follows the only path leading to the liver, the large and small splanchnic branches of the sympathetic.

Certain pathological alterations of the nervous system, of which I will speak immediately, have had the same result. A Rouen physician, M. Leudet, has ascertained that diabetes and glycosuria are produced in certain affections of the encephalon. Glycosuria has been known to be the consequence of a fall on the head. Another process is the administration of some poisonous substance; as woorara, for example. The poisoned animal becomes diabetic. Morphia in large doses will produce the same effect.

In all these cases, if the viscera of the animal be examined, it is seen that the circulation is considerably increased there. Bernard's expression is—'The hepatic cells, foci of glycogenic matter, find themselves surrounded by a kind of sanguineous network. The

circulation becoming more active in this network, the contact of the sanguineous fluid with the cellular fluids is better assured, the action on the glycogenic matter is more energetic, the transformation becomes more abundant, and the sugar produced is immediately carried away. The increase in the rapidity of the circulation of the liver increases the glycæmia.' This is the theory of the operation. Still, this increase in the circulation of the liver is not the result of paralysis of the vaso-motor nerves. The floor of the fourth ventricle is certainly one of the most clearly defined of the vaso-motor centres, and diabetes, caused by woorara or by morphia, is produced not by any direct influence of these poisons on the vessels of the liver, but through the medium of the nervous system, precisely as in artificial diabetes from puncture of the floor of the fourth ventricle. Thus, if it were by means of vaso-motor paralysis, the functional trouble would persist as long as the lesion ; whereas it is not so. The diabetes is essentially temporary. It is by nervous excitation which is temporary. The instantaneous action of the fourth ventricle and the resultant irritation are the causes of the circulatory superactivity and of the diabetes. The effect persists as long as the irritation. Bernard believes that the phenomenon may be referred to the momentary excitement of the medulla oblongata produced by the puncture. The first action, too, of woorara and morphia on the nerves is an irritation of their peripheral or central extremities. The effect of various pathological lesions is similar. A lesion of the medulla oblongata or of the upper part of the

spinal cord sufficient to cause irritation and excitation, and not paralysis, will be likely to produce diabetes. Cyon and Aladoff corroborate Eckhard's statement—that diabetes occurs in dogs an hour after the section of the inferior cervical or upper dorsal ganglia.

What, then, are the pathological conditions that have this direct influence on the causation of diabetes? Congestion of the floor of the fourth ventricle near the calamus scriptorius, blows on the occiput, falls on the head, fracture of the cranium, or traumatic injuries of the brain, apoplexy, with intracranial effusion, intracranial tumours, are some of the most important. Dr. Roberts observes that it is probable that in all the traumatic cases the injury implicated some part of the nervous system. Luys describes a case of a diabetic in whom lesion of the anterior wall of the fourth ventricle was found; and in connection with this point an observation of Dr. Richardson is interesting. He found the symptoms of a diabetic painter much increased whenever he, as a house painter, had to look constantly upwards, with the head thrown back, as in painting a ceiling. Several cases, too, have been recorded where diabetes has co-existed with the presence of tender spots in the back of the neck, and the diabetes has vanished with the tenderness on the application of blisters, cupping, or setons to the neck.

Dr. Ogle mentions that intermittent diabetes has been said to have followed traumatic and other varieties of lesion of the brain, and other parts of the nervous system, including non-traumatic extravasation of blood, a calcareous growth projecting from the basilar groove

of the occipital bone, pressing on the pons, as also certain general commotions of the nervous system, mental and emotional excitement, even old age, disturbance of the functions of the liver and lungs, inhalation of chloroform, affections of the skin and superficial parts, such as boils and carbuncle, and certain unwonted states of the blood, temporary fever, attacks of a convulsive character, such as eclampsia, hysteria, the use of certain articles of food or of medicine.

Dompeling has translated from the Dutch a case of diabetes, caused by a tumour (sarcoma fasciculatum) of the size of a nut in the right half of the medulla oblongata, producing partial atrophy of the right vagus and spinal accessory nerves, partial paralysis of the internal muscles of the right eye, and persistent diabetes, with death occurring in six years. A very interesting case in which diabetes mellitus supervened upon a severe injury to the heart, in an infant of eight months, is recorded by Dr. Rossbach, of Herbsleben, in the 'Berliner Klinische Wochenschrift' for June 1874. The child fell from the nurse's arms; a convulsion immediately came on, followed by unconsciousness, vomiting, and other symptoms of shock; depression of the fontanelles, and dilatation of the pupils. No fracture of the skull could be discerned. Reaction began on the third day, and in a few days the child had almost recovered. Four weeks after the child was found emaciated and ravenous, and the urine, which was passed in large quantities, found to contain sugar.

Much more minute investigations have, however, been undertaken in England on this question.

Dr. Dickenson brought a series of cases before the Medico Chirurgical Society, and showed that peculiar morbid changes were constantly found in the cerebro-spinal system.

In all, the alterations were of the same nature and in similar situations. The earliest alteration recognised consisted in a dilatation of the blood-vessels, particularly of the arteries, with accumulation and frequent extravasation of their contents. The next was a degeneration of the nervous matter at certain points outside the swollen vessels, probably caused by the intrusion into it of blood corpuscles. The degenerative process occasioned destruction and excavation of the tissue round the vessel. Cavities were thus produced, often large enough to be striking objects, even without the microscope; cavities which contained blood-vessels, extravasated blood, grains of pigment, and the products of nervous decay. Finally, the contents appeared to have become absorbed, so that simple vacuities were left. The perivascular sheath was variously stretched and altered in character, and became loaded with pigment; but it seemed that these alterations were consequent upon the dilatation of the vessel, extravasation of blood, and excavation of nervous matter. As to their situations, the changes occurred in constant association with arteries. They were found in every part of the spinal cord and encephalon, attaining their greatest development in the medulla oblongata and pons Varolii. The excavations were generally the most marked where the blood-vessels piercing the brain were the largest and most numerous. They were frequently in connection

with folds of the pia mater. The regions affected with the greatest frequency were the olivary bodies, the vicinity of the median plane of the medulla, the grey matter of the floor of the fourth ventricle, a fissure just internal to the origin of the facial nerve which lodges a process of the pia mater, and a depression similarly occupied, which penetrates from between the anterior crura towards the centre of the pons Varolii. The optic thalami and corpora striata were involved, though to a comparatively slight extent. The septum of the ventricles and the white matter of the convolutions displayed the alterations in a remarkable manner. The changes especially affected the white matter, though the grey matter at the floor of the fourth ventricle and of the spinal cord are exceptions to this statement.

In the cord the most conspicuous change was the enlargement of the central canal, probably connected with the degeneration of tissue, of which many evidences were found there and elsewhere. The nerve-cells of the brain and cord were generally perfect. Such parts of the sympathetic system as were examined, namely, the upper cervical and semi-lunar ganglia, were apparently natural.

Dr. Dickenson thinks that the nervous changes are not the result of the state of the blood, because, though the arteries are diseased, the veins and capillaries are not; and because, although the blood traverses the whole body, no analogous failure in nutrition is produced in any other organ; and he believes the nervous changes are primary, first, because the changes in the brain are in their nature and situation such as

physiology has shown to be capable of producing that symptom ; secondly, because alterations similar in kind, though differing in distribution, occur as belonging exclusively to the nervous system, quite independently of diabetes.

In a sixth case Dr. Dickenson found similar lesions in the cord, in one olivary body, and in the corpora striata, but in addition to these there was an ovoid channel in the white matter of the cerebellum partly filled with the globular débris of nerve-structure.

In a seventh case, where the patient died at a very early period of diabetes, there were similar lesions in the medulla oblongata, and punctiform hæmorrhages of the pons, whilst in the white matter of the cerebellum, and especially in the central part of the corpus dentatum, large vessels packed with corpuscles lay in eroded channels.

Dr. John Ogle gives some cases of great interest with reference to the connection of diabetes and abnormalities of the nervous system.

In his first case the diabetes of old standing disappeared coincidently with the appearance of some paralytic symptoms, which increased and persisted up to the death of the patient. The scalp and the bones of the skull presented nothing unusual. The dura mater was considerably adherent to the skull generally, but was itself healthy. There was much dark staining of the cerebral membranes along the track of the blood-vessels. The surface of the brain presented no unnatural appearances ; but on section it proved to be what is termed a wet brain. The anterior portion

of the middle lobe of the brain, on the right side, was to a very great extent exceedingly softened, but not in any degree reddened or otherwise discoloured. On examining the arteries at the base of the brain, the middle cerebral one on the right side and in the neighbourhood of the softened portion of brain was found to be plugged up with a mass of firmish fibrine. The coats of the vessel did not appear to be affected. Other parts of this hemisphere of the brain were natural. The corresponding portion of the middle lobe of the left side of the brain was also, but to a very slight degree, softened, and the middle cerebral artery occupied by a small plug. Dr. Ogle thinks that the nerve lesion was secondary in point of time to this diabetes. It is doubtful if this was so. More probably the lesion of the nerve-centre at an early period only gave rise to excitation, and diabetes was induced; when it advanced so far as to cause paralysis, excitation ceased and the diabetic symptoms vanished. Many diabetic patients die paralytic or comatose. In 15 fatal cases recorded in the St. George's Hospital Reports there was no account of any nerve lesion except in the last case, where Lockhart Clarke examined the medulla. It is worthy of remark that this was the only one of the 15 in which a microscopical examination was made. To the naked eye the whole of the medulla appeared perfectly healthy; but on examining these sections under the microscope, it was evident that the fourth ventricle, from the point of the calamus scriptorius upwards for about a quarter of an inch, was the seat of finely granular deposit, extending through the epithe-

lium for some distance down the raphè, in which, as well as in the substance of the medulla on each side of it, numerous corpora amylacea were interspersed. In no other part of the medulla, nor in the third ventricle, could he perceive any abnormal appearance. The central part of the pons seemed rather more vascular than usual; but this might be accidental. It is more than probable that some lesions would have been found in other cases had they been sought for. There are at least two varieties of diabetes, one so far remittent that the sugar disappears from the urine when all saccharine and amylaceous articles of food are removed; whilst in the other the sugar remains under any diet. It is probable that in the former the irritation is of a temporary nature—in the latter the lesions which we have been describing are present.

Consult—

- Wilks, 'Path. Anatomy.'
- 'Med.-Chir. Trans.,' 53. Dr. Dickenson.
- 'Med. Times and Gazette,' March 8, 1862. Dr. Richardson.
- 'Med. Times and Gazette,' June 20, 1874.
- 'Med. Record,' 1, 692. Dr. Claude Bernard.
- 'St. George Hosp. Rep.,' vol. i. Dr. Ogle; and notes of cases by Pavy, Noble, Tardieu, Luys, Roberts, Griesinger, Richardson, Kunkler, Buttura, Abeille, and Goolden.

LECTURE XIV.

OPHTHALMOSCOPY IN DISEASES OF THE NERVOUS
SYSTEM.

LESIONS of the retina and optic nerve are not only important as accounting for amblyopia and amaurosis, but as evidences of various abnormalities in the brain or membranes.

Speaking roughly, we meet with four chief lesions of the retina—choked discs (called by Dr. Allbutt *ischæmia papillæ*), hæmorrhage, optic neuritis, and atrophy.

I need not say that it is impossible to learn much of the ophthalmoscope in a lecture. It is only by very constant use of the instrument that any adequate knowledge can be arrived at. Still it may be possible to point out to you some of the lesions observed, and to tell you of the morbid circumstances, other than retinal, under which these lesions have been found.

Observations have been made on the retina in the course of the following diseases or abnormal states:—Meningitis, encephalitis, cerebral softening, cerebral hæmorrhage, phlebitis of sinuses, chronic hydrocephalus, cerebral tumour in various situations within the cranium, cerebral sclerosis, compression of brain and fracture of

skull, acute insanity, dementia, idiocy, general paralysis, epilepsy, chorea, chronic myelitis, various injuries to the spine and cord, locomotor ataxy, syphilis, leukæmia, albuminuria, diabetes, delirium in acute diseases, toxæmia from various causes, paralysis of the third pair, embolism of the central artery of the retina, and diphtheria. It will be convenient to take the most important of these in order.

First, then, meningitis. By the mirror alone it is generally impossible to decide whether we have to deal with tuberculous meningitis or not. In a few very rare cases tubercle has been found in the retina, and has even been observed during life by the aid of the mirror. But it is wise to realise what an instrument like this will not do, as well as what it will do. It does not save, it is not meant to save, the practitioner from taking all the symptoms and history of the patient into careful consideration. It will help in two ways; it will show conditions of meningitis of which the symptoms are so slight that the disease is not easily recognised by them; and it will teach the positions of the meningitis. It will tell us very little of meningitis of the convexity, very little of meningitis at the most posterior portion of the base, but it is a delicate test for meningitis of the base affecting the anterior portions of the brain, and persisting for a certain time.

In acute idiopathic meningitis, in tuberculous and in rheumatismal meningitis, the disturbance of the cerebral circulation is almost identical, and therefore very similar vascular lesions are produced mechanically at the fundus of the eye; but we may remember that

meningitis of the anterior portion of the base is much more likely to be of the tuberculous form than any other. The cerebral hyperæmia, the effusion into the ventricles, the engorgement and thrombosis of the meningeal veins, the repletion of the cerebral sinuses, especially the cavernous, obstruct the venous circulation of the eye in meningitis of the base, may retain the blood in the veins of the retina, and so produce intra-ocular lesions, which are known by the name of choked discs or ischæmia papillæ. Besides this, the inflammation seems often to creep down the optic nerves, causing optic neuritis, and this lesion is very often associated with ischæmia papillæ.

Further, also, the sheath of the nerve may be implicated in the inflammatory changes, and perineuritis be the result.

Ischæmia papillæ then is due to mechanical causes :

1. Pressure on the cavernous sinus chiefly, causing a diminution or an arrest of the flow of venous blood, and so a great congestion of the optic nerve and escape of the vascular contents, either serum only, or small ecchymoses as well.

2. This condition is partly caused, and always intensified, by partial strangulation of vessels by the sclerotic ring, a necessary result of the anatomical distribution of the parts.

‘In ischæmia papillæ (quoting Dr. Allbutt) the trunk of the nerve is unchanged, and all the morbid signs are confined to its intra-ocular termination. This part is greatly swollen, and it generally rises steeply on one side, and sinks gradually to the level on

the other. There is some swelling also of the fibres themselves, so that they lose their transparency. The colour of the papillæ is often a mixture of dirty grey and red, due to the mingling of passive effusions with distended capillaries and hæmorrhages, but in other cases there is not much extravasation of blood, and the protruded disc looks bright or almost transparent.' The veins of the retina are enlarged and generally very tortuous.

Von Gräfe's account of this lesion in the first case he observed was that the optic papilla was considerably and irregularly swollen in such a manner that it rose abruptly on one side, and on the other sloped down almost imperceptibly to its ordinary level. The normal transparency of its tissue had disappeared, giving place to a greyish tint, with a very strong shade of red; the adjacent portions of the retina exhibited exactly the same change, so that the choroidal margin of the papilla was hidden from view. The retinal opacity was uniform, presenting, nevertheless, with the erect image a slightly striated aspect, which followed the course of the optic nerve fibres. The retinal veins were larger than normal, extremely tortuous, very dark-coloured in parts, and issued in a very irregular manner from the opaque tissue; the arteries were relatively diminished in size. The opacity of the retina progressively decreased from the border of the papilla.

Of course any pressure in the same situation may lead to this condition of choked discs, and existing by itself it is much more commonly found as the result

of cerebral tumour than of meningitis. Here at once is seen the necessity of taking all the symptoms of a case into consideration. But it may exist as the main optic phenomenon even in meningitis. Commonly, however, we find associated with it true optic neuritis, in which the inflammation has crept down the nerve from the base of the brain; it is often called 'neuritis descendens.' In this form the disc is also swollen, but not to the same extent, and it does not rise abruptly on one side; it is of a greyer colour, at the most of a reddish tinge; but it never presents the intense red of the other form. Moreover, the morbid process, which, as a rule, seems to develop more gradually, extends to a much greater distance beyond the disc, and attacks all the layers of the retina, where it may manifest its presence by white patches, not to speak of numerous hæmorrhages.

No doubt Dr. Allbutt is right in saying that the optic neuritis may not be seen by the mirror in all cases of tuberculous meningitis, because the neuritis may not have had time to reach the discs; but that it is most common in those meningeal inflammations which, like the syphilitic, have the favourable conditions of contiguity, duration, and activity.

An interesting illustration of this point is seen in a case reported in *Medical Times and Gazette*, June 17, 1874.

The case was one of tuberculous meningitis, running a rapid course of only seven days after the first well-marked symptoms.

In this case there was an entire absence of affection

of the eye as seen with the ophthalmoscope. I have myself, on several occasions, realised the same thing in early stages of tuberculous meningitis.

In those cases in which there has been much proliferation of the interstitial connective tissue of the nerve there results atrophy of the nerve from the compression of the nerve-fibres by the connective tissue. The appearance of the disc in this condition will vary according to the stage of the disease and the amount of the compression. Atrophy, however, is a change that demands time, and is therefore a phenomenon seen in chronic meningitis rather than in acute.

Although there is no certain rule on the subject, yet in many cases the vascular lesions at the fundus of the eye are more marked in the eye that corresponds to the cerebral hemisphere where the inflammation is most intense and the obstruction to the circulation most considerable.

It is thought by Bouchut and others that children may get well of acute meningitis, and that in them there are often persistent disturbances of vision, depending on the alteration of the media and the membranes of the eye, consequent on the vascular lesions occasioned by inflammation of the meninges. It is more than probable that many children recover from meningitis when the inflammation is very subacute, even from meningitis of a tuberculous form. Such cases, however, never, I believe, present decidedly acute symptoms. Drs. Allbutt and Crichton Browne have recognised in many non-congenital idiots optic changes which are precisely those that are found in persons who have

had meningitis. In fact, it is partly from ophthalmoscopic observations that the diagnosis of the occasional causation of idiocy by meningitis has been made.

In the chronic meningitis, so often met with in drunkards, the convexity of the brain is usually attacked, and no retinal changes are met with.

In syphilitic meningitis of the base we may meet also with what is called primary atrophy, in which the distal parts of the nerve have been separated from the central, and we find dwindling of the vessels, and either a diminution of the disc and a tendency to cupping, or a very white disc in which little is found but connective tissue.

Encephalitis.—Partial encephalitis is never sufficiently extensive or intense to act mechanically on the circulation at the fundus of the eye; and it is by destroying the cerebral pulp at the origin of the sensors and motor nerves of the eye that it causes different alterations of vision.

It stands to reason, therefore, that we find atrophy as the lesion at the fundus of the eye, atrophy from separation of the distal from the central end of the nerve. There is rarely torsion or dilatation of the veins of the retina, never hæmorrhage of the retina, almost always amaurosis from atrophy. I need not say that this lesion will only be met with when disorganising encephalitis attacks the corpora quadrigemina.

Bouchut says that inflammation of the optic nerve or the neurilemma may be propagated to the brain in an ascending course, and is often the cause of a chronic partial encephalitis, occupying the corpora striata, the optic thalami, and the corpora geniculata.

It is probable that in general encephalitis there is great suffusion of the retina, such as has been seen in the eyes of patients immediately after a paroxysm of mania. Facts, however, are wanting in support of this statement.

Cerebral Softening.—Softening of the brain seldom, if ever, by itself causes any alteration that can be detected by the mirror. It cannot induce any obstruction to the flow of blood from the retina, nor can it cause neuritis. Softening, however, of the optic centres will cause atrophy; and cerebral softening itself often depends upon disease of the cerebral vessels, disease which, even if it does not affect the vessels of the optic centres, may attack the arteria centralis retinae, causing first anæmia and then atrophy of the disc. With the exception, therefore, of the cases in which softening attacks the optic centres, it is right to say that atrophy of the disc coexisting with cerebral softening owns no connection further than a similarity of cause in arterial degeneration.

Cerebral Hæmorrhage.—The condition of the retina in cerebral hæmorrhage is somewhat complicated, because hæmorrhage of the brain so often depends upon renal disease, and renal disease frequently produces retinal changes, of which I shall speak by-and-by. Apart, however, from these albuminuric alterations in the eye, cerebral hæmorrhage seldom leads to any change in the fundus of the eye; but optic neuritis is occasionally a sequence of it. Bouchut thinks, however, that large cerebral hæmorrhages may cause optic lesions by the obstruction they produce to the return

of venous blood from the eye, by compression, in the same way as cerebral tumour; such cases are rare. If this compression from cerebral hæmorrhage occurs, we shall have choked discs.

I cannot agree with him when he says that in cerebral hæmorrhage there is sometimes produced an atrophy of the papilla, which is the ultimate consequence of irritation of the optic nerve by the serous infiltration of which it had been for a time the seat. Atrophy is never produced by these means, even if serous infiltration is ever one element among many in its causation.

It should be remembered in connection with this subject that hæmorrhage of the retina may be met with independent of cerebral hæmorrhage and of albuminuric retinitis; that it may be due to backward pressure upon the capillaries, or to rupture of a minute aneurism either in cases in which there are many aneurisms (microscopic) of the pia mater or of the vessels of the cortex, or independently of aneurism elsewhere.

Phlebitis, or Thrombosis of Sinuses.—In this morbid state we see very intensely the influence of obstructed venous circulation. With phlebitis of the sinuses of the dura mater, or indeed with the formation of coagula in them from causes other than phlebitis, we may have, according to the sinuses affected, interference with, if not absolute obliteration of, the cavernous sinus, hydrophthalmia, and congestion of the choroid, dilatation, tortion, and thrombosis of the veins of the retina, and choked discs.

Chronic Hydrocephalus.—The changes in the fundus of the eye in chronic hydrocephalus are twofold; first, the choked discs, &c., mentioned as occurring from obstructed sinuses, and in this case caused by the pressure of the fluid in the distended ventricles on the cavernous sinus, with the additional strangulation by the sclerotic ring of the already congested vessels, with sometimes hæmorrhages and exudations consequent on the congestion; and secondly, the pressure of the fluid on the optic chiasma may produce atrophy of the optic nerve, and therefore of the optic disc. Under such circumstances the pressure has probably been from the fluid accumulated in the third ventricle.

Cerebral Tumour.—Alterations in the retina of very various kinds may depend on the presence of cerebral tumour. No doubt the rule is that cerebral tumour causes ischæmia papillæ. But great variations in the lesion occur according to the position of the tumour, according as it has interfered directly with the optic centres, according as there is any meningitis round it, and according as it may or may not press on the optic nerves and chiasma.

Taking then our three main varieties of lesion, we find tumour causing choked disc by interfering with the venous ebb from the eye; optic neuritis only, if there is meningitis associated with the tumour, and depending, therefore, not on the tumour directly, but on the meningitis; atrophy, by pressure of the tumour on the optic nerve, of the tumour or of the hydrocephalus secondarily induced by it on the optic centres or tracts, by softening around tumours, such softening implicating

the optic centres, and lastly by the propagation of sclerosis.

It is easy to understand that a tumour far back in the encephalon, by interfering with the venous flow through the venæ Galeni, may produce hydrocephalic distension of the ventricles, the effect of which on the circulation of the eye would be the same as that of hydrocephalus produced by any other cause.

Dr. Allbutt has passed in review the effects of the modes of action of tumours situated in various portions of the brain. It is not possible in these few lines to do adequate justice to the beauty and accuracy of his observations and reasoning. Briefly, however, it may be stated that tumours of all the lobes of the cerebral hemispheres induce optic phenomena by their interference with the basilar portion of the brain, either by direct pressure on the cavernous or petrosal sinuses or on the optic nerves, tracts, or centres, or by leading to softening of the same important parts, or by setting up meningitis of the base, which will lead to neuritis descendens, or by indirect pressure on the same part induced by hydrocephalic distension of the ventricles, from interference with the ebb of blood from the venæ Galeni by a tumour obstructing these vessels in their course.

There are no optic phenomena when tumour is confined to the corpus callosum; and these appearances are often also absent in tumours of the optic thalami. Here incidentally we have another proof in addition to many afforded us by pathology that the optic thalami have nothing to do with sight.

Tumours of the crura cerebri are so close to the

great visual centres or tracts that it is probable they would cause atrophy by direct pressure. Facts, however, are wanting. The oculo-motorius is almost invariably affected, and in some of the few cases on record amblyopia or amaurosis has been observed.

Tumour of the cerebellum has no direct action on the eye, but indirectly it may cause choked disc, or in another way atrophy. 'Thus pressure on the occipital bone, such as would be exercised by tumour, means pressure upon the lateral sinuses, and pressure upon the tentorium also means pressure upon the straight sinus, upon the outlet of the veins of Galen, and upon the Torcular Herophili itself. Slight enlargements, therefore, of the cerebellum must very soon interfere seriously with the reflux of venous blood. Again it is clear that in enlargement of the cerebellum, especially of its median and anterior parts, there must be a resolution of the pressure in the direction of the meso-cephalon, as this is the direction of the least resistance.' From this cause the corpora quadrigemina may be very easily disorganised, and atrophy of the optic nerve would be the result. Or softening may proceed from the circumference of the tumour along the processus ad testes to the corpora quadrigemina, and the optic nerves will waste, either as a consequence of the destruction of the visual centres, or because they are themselves compressed.

Tumours of the crura cerebelli cause distension of ventricles by fluid, and thereby choked discs.

Tumours of the corpora quadrigemina lead to amaurosis and progressive atrophy of the optic nerves. Tumour of the pons may cause a twofold lesion;

choked discs by the pressure of hydrocephalic fluid, and atrophy from softening or destruction of the corpora quadrigemina.

We have no examples of tumour of the medulla oblongata, but Dr. Allbutt says: 'It is unlikely that tumours of the bulb are attended with amaurosis, unless they are complicated with sclerosis or meningitis, or are large enough to involve the encephalic centres above.'

Tumours of the anterior fossa cause amaurosis pretty constantly; they may act in three ways: either by direct injury to the nerve, severing it from its central attachments, and so causing atrophy; or by retarding the reflux of blood, and thus choking the discs; or by irritation of the connective elements of the nerves, with consequent neuro-retinitis.

Tumours of the middle fossa may cause atrophy, if the nerve is compressed; choked discs or neuro-retinitis if meningitis is present.

Tumours of the posterior fossa may induce choked discs indirectly by setting up hydrocephalic distension of the ventricles, or by meningitis; or they may by means of the meningitis lead to optic neuritis.

Neither the aid of the mirror nor any physical sign or symptom will inform us with moderate certainty of the nature of the tumour. It can only be said that, as a rule, aneurisms interfere less than other tumours with the special senses, and that therefore intracranial aneurism does not often cause any direct abnormality in the fundus of the eye. No doubt this is due to the less density of these tumours.

More or less intense congestion of the retina, partial or general œdema round the papilla, dilatation and torsion of the veins of the retina, in a patient who has lost consciousness after a wounding of the brain, indicate a severe contusion or a considerable compression of the brain.

There is no specific lesion diagnostic of fracture, unless fracture of the base injure the optic nerve or cause basilar meningitis. Retinal hæmorrhage may be met with after fracture.

Acute Insanity.—In acute delirium there is generally congestion of the vessels only. As far as we may judge from the inherent difficulties in making accurate observations in acute mania, or even in acute melancholia, there is no very special lesion found in the retina. It is certain that, even in cases in which there is evident cerebral hyperæmia, the circulation of the retina is full in no proportionate degree. Bouchut says there are no optic lesions peculiar to folie. Still some such hyperæmia has been found after the paroxysm of acute insanity has passed over. Dr. Noyes found in acute and subacute mania hyperæmia of nerve and retina in fourteen out of twenty cases, and the eye normal in six.

Chronic Mania, however, is frequently connected, as we have seen, with absolute persistent lesion, and the changes at the fundus of the eye bear a due relation to the amount, the nature, and the position of these changes. We may have choked discs from interference with the nervous reflux, or neuritis from extension to the optic nerve of basilar meningitis, or atrophy as the result of this latter condition, or as a consequence of

sclerosis of the nerve. Dr. Noyes found in three cases out of six of chronic mania hyperæmia or signs of inflammatory action.

In *Dementia* the appearances vary somewhat according to the stage of the disease. In 12 out of 18 cases observed by Dr. Noyes there were found hyperæmia and infiltration of the optic nerve and retina, the vascularity affecting chiefly the venous and capillary circulation. In late stages of the disease, however, where amaurosis exists, it is due to atrophy of the discs.

In *General Paralysis* of the insane a very similar remark may be made. In early stages hyperæmia and infiltration of the nerve and retina may be found. Dr. Noyes says, the striation of the retina near the nerve is often extremely pronounced, rendering occasionally the edge of the nerve hazy and indistinct. But the specific feature of such cases later on is atrophy of the nerve, beginning, Dr. Allbutt thinks, at the end of the first stage or the commencement of the second, and proceeding to amaurosis. The proper nerve-structure vanishes in consequence of pressure by a hyperplasia of connective tissue. Dr. Aldridge thinks this condition is most complete in females, and is generally most pronounced in the left eye.

In *Epilepsy* the results recorded by various observers do not completely agree. Bouchut and Allbutt consider that in the intervals between the fits there is no lesion, and this would be in accordance with my own experience. I understand Dr. Aldridge to say, however, that in his cases there have been generally found

hyperæmia of retina and optic disc with tortuosity of veins. He finds also during the convulsive stage injections of the optic discs, with the arteries larger than usual; whilst Dr. Allbutt has seen at the same period anæmia of the discs corresponding to what is believed to be the state of arterial contraction and consequent anæmia within the brain, and hyperæmia in a similar number of cases. Dr. Hughlings Jackson has several times found anæmia of the discs in the epileptic state.

All, however, agree that there is a congested appearance of the discs when the fit is over, especially a dilatation of the veins of the retina, which often will persist for some days, and diminish only very gradually. Coincident with this, and also continuing for a variable period, is a general anæmia of the retina. Optic neuritis, choked discs, and atrophy are rare in epilepsy, but Bouchut asserts that there are often in epilepsy atrophies of the choroid, inflammation of the retina, with deposit of pigment, exudations on the retina, and hæmorrhages of it, which evidently depend on this disease.

In *Chorea* there is usually no change; but Dr. Jackson speaks of the optic discs being badly margined, with large and wavy veins, and hyperæmia of disc in this disease.

Diseases of the Spinal Cord are sometimes accompanied by lesions of the optic nerve or retina. In locomotor ataxy, for instance, these abnormalities are found, and are variously explained by different authors. Bouchut states that in almost all cases of locomotor

ataxy there are produced disturbances of vision, cloudiness, strabismus, diplopia, and amaurosis, which are in relation with the lesions of the columns of the cord, and reveal the existence of this alteration; and he believes the connection between the optic and motor phenomena to be carried out by means of impairment of the power of the sympathetic in the neck, so closely associated with the columns of the cord.

This idea, however, has been quite disproved. The connection between the optic and motor phenomena is simply a similarity in the nature of the lesion; the appearance in the eye is atrophy of disc caused by sclerosis of the nerve, just as the want of motor co-ordination is caused by sclerosis of part of the cord, mainly the posterior columns. There is no direct connection.

In certain cases of injury of the spine, especially in its upper portion, in meningitis of the spinal cord, in chronic myelitis, always after the disease of the spine or of the cord has persisted for some time, there may be found hyperæmia of the fundus of the eye, due in all cases to the extension upwards along the base of the brain of the inflammation of the meninges.

Chronic myelitis, if it ever does exist without meningitis, would not cause this hyperæmic change. Injury to spine, if only it existed *per se*, and did not involve the spinal meninges, and by extension of mischief upwards the cerebral meninges, would have no effect on the retina. These optic lesions are due solely to the creeping upwards of a meningitis that was originally spinal, but ends in being cerebral.

We have already mentioned that atrophy of the nerve is sometimes found in syphilitic patients, and is a sequence of slow creeping meningitis, a neuro-retinitis being set up, and atrophy being a later result. A lesion appearing early in the history of syphilitic patients is retinitis, in which we see hyperæmia of the retina, effusion of serum into its nervous texture, with a grey hazy look, especially, as Mr. Oglesby says, near the macula lutea. In marked cases there may be great tortuosity of the veins.

Leukæmia.—General pallor of the retina and choroidal vessels and of the papilla is the characteristic feature, according to Allbutt, of leukæmia. He speaks also of a striated cloudiness of the retina in the neighbourhood of the papilla and irregular spots near the macula lutea, with a number of glistening white round spots at the periphery.

In Albuminuria we find various lesions of the fundus of the eye. These are much more usually met with in the small granular kidney than in any other form of renal disease, but they have been seen associated with the large kidney of chronic tubal nephritis. The lesions are twofold; either a mere amaurosis, existing with uræmia, and due most probably to anæmia of the cerebral vessels, and without retinal change, except, maybe, some anæmia both of retina and disc; or the true albuminuric retinitis, which is never an early symptom of the disease, and is evidenced at first by infiltration of the disc, with great vascularity, and later on by spots of degeneration, these being whitish spots or masses, and extravasation of blood. Virchow

thinks these white spots are merely decolourised clots, but it is probable that Dr. Allbutt is right in denying this. The spots and hæmorrhages seem to have a special proclivity for the surroundings of the disc and the macula.

Liebreich's account of his beautiful plate of albuminuric retinitis is that the greyish cloudiness of the papilla and its immediate neighbourhood is caused by serous infiltration and augmentation of the cellular tissue of the retina. The white opaque zone which surrounds this portion of the fundus is produced by the sclerosis of the nerve-fibres and fatty degeneration of the cellular tissue. The latter occurs isolated in the very small round spots which are on the edge of the white figure, and which assume a star-like arrangement at the macula lutea, looking as if sprinkled upon the fundus. The numerous hæmorrhagic extravasations nearly all lie in the most internal layer of the retina; the striation of the spot is produced by the arrangement of the blood corpuscles in rows between the nerve-bundles; and everywhere the direction of the striæ corresponds with the course of the nerve-fibres. In other cases there were also round extravasations situated in the deeper layers of the retina, and other very extensive ones situated between the retina and the choroid. The veins may be overfilled, the arteries almost empty.

Bouchut says: 'A more or less extensive serous infiltration of the papilla, linear hæmorrhages along the vessels, spots of hæmorrhage, or white fatty patches on the retina, sometimes, in fine, a fatty infiltration of

the band of the optic nerves extending to the tubercula quadrigemina, are the causes of the amaurosis, which shows itself at the beginning and in the course of acute and chronic albuminuria.'

In Diabetes atrophy of the discs may be met with; the serous infiltration of the papilla or of some points of the retina, the small hæmorrhages, and white fatty granulations of the retina occasionally found, are probably due to the coincidence of albuminuria with diabetes.

In Embolism of the arteria centralis retinæ there is anæmia of the disc and diminution in size of all the vessels, but very specially of the arterial branches, which may be extremely small.

In convalescence from acute disease there may be amaurosis, or at least some interference with perfect vision, due either to anæmia of the retina, consequent on general anæmia, or to various lesions, Ischæmia, or optic neuritis, depending on a low form of meningitis that has occurred in the previous disease, even if this previous disease has not been accompanied by renal changes sufficient to cause albuminuric retinitis. This latter lesion is often doubtless the cause of amaurosis occurring during convalescence from diphtheria.

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Liebreich, 'Atlas of Ophthalmoscopy.'

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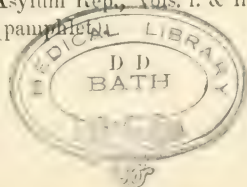
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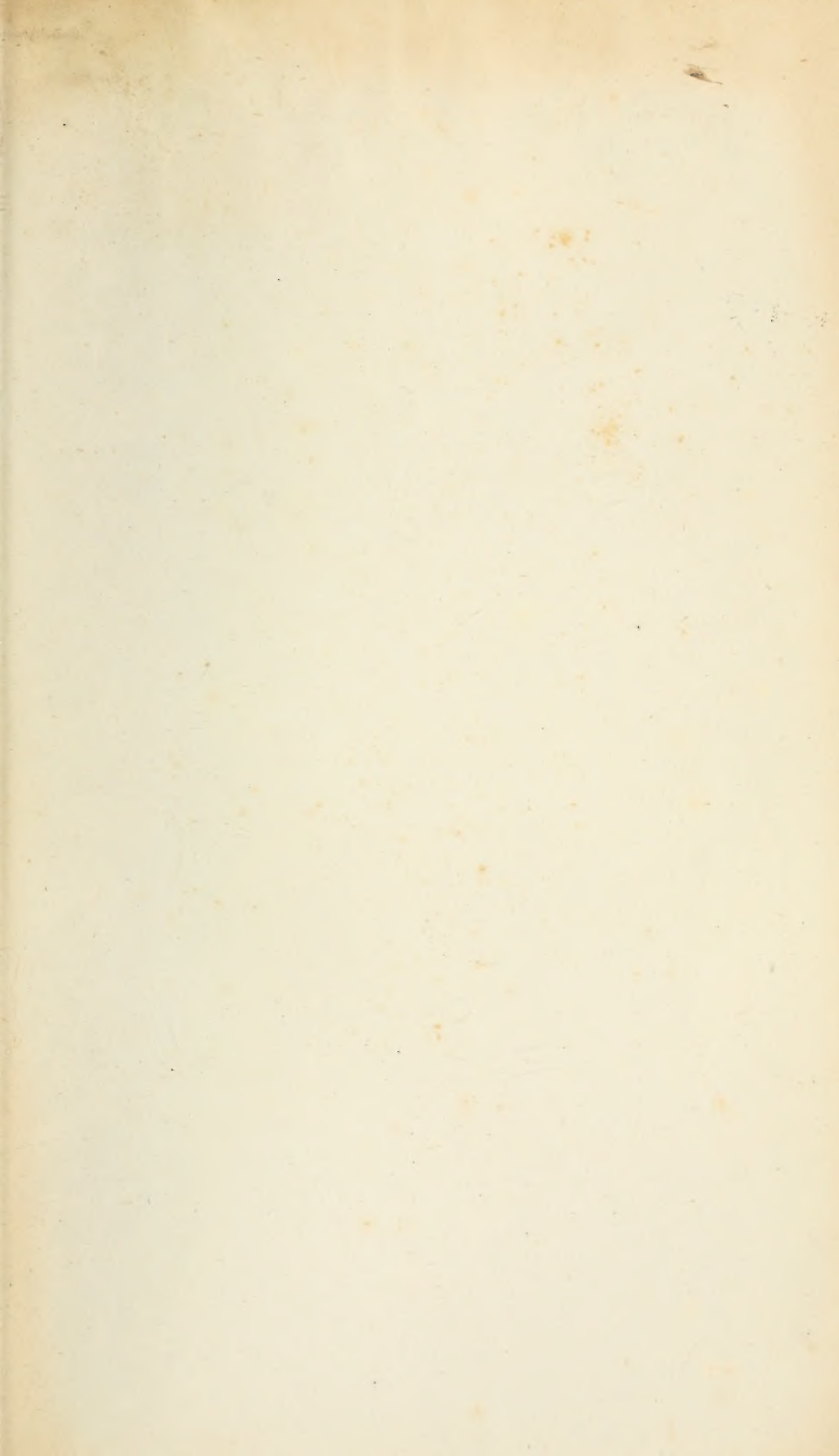
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